The Manitoba Medical Review

Vol. 40 MAY, 1960 No. 5

Foreword

This issue of the Manitoba Medical Review is designed to demonstrate, through case presentation and clinical reports by members of their Active and Consulting Staffs, some of the activities of the Winnipeg Municipal Hospitals and some of the services offered to the medical practitioners and their patients. The relative success of the operation of these Hospitals to date is due, in no small measure, to the interest and support which have been received from the medical profession at large.

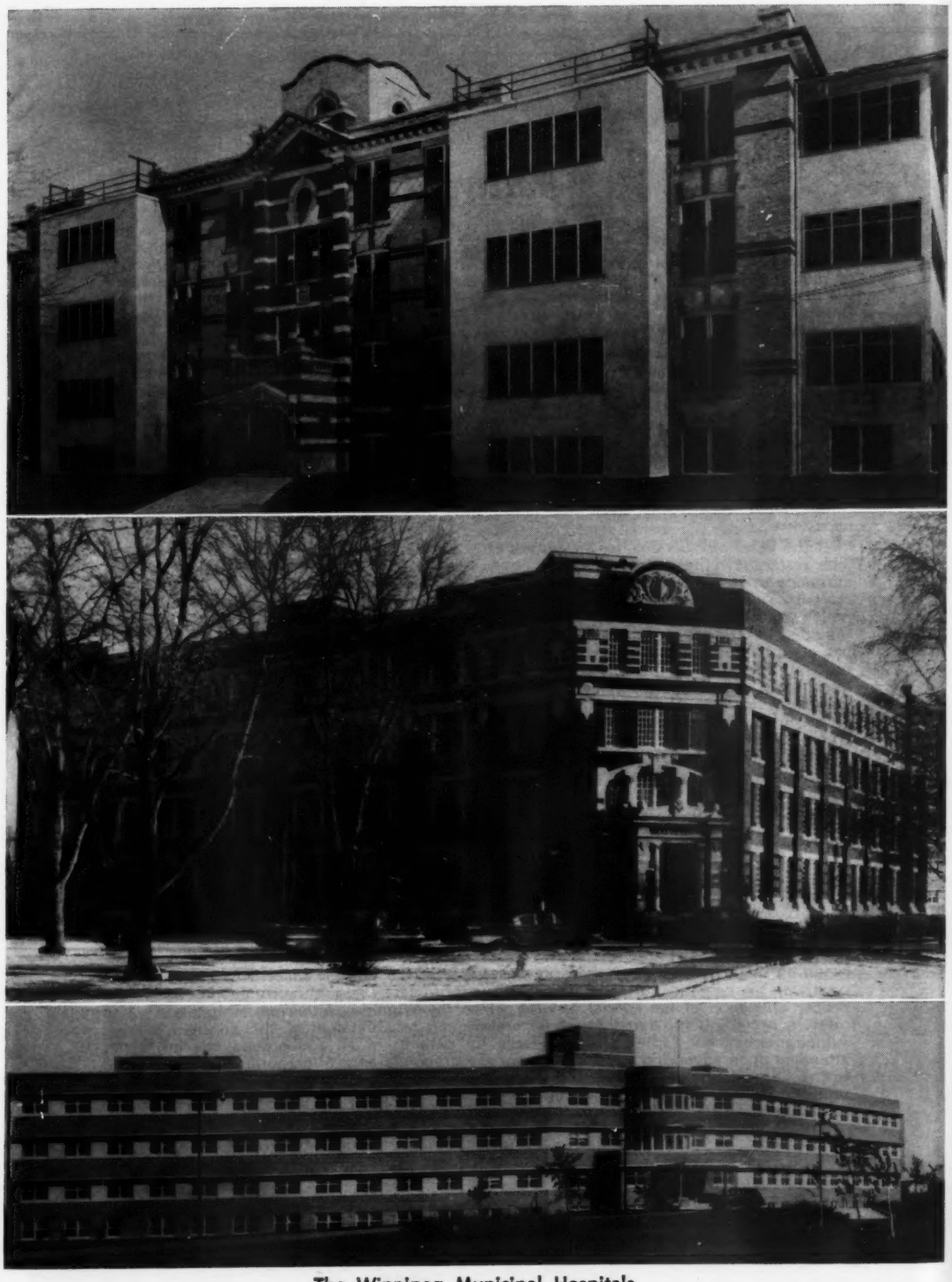
Although built to serve specific needs, the role of these Hospitals gradually changed with the changing needs for hospital accommodation. At the beginning, the need was for the care, treatment and isolation of infectious disease. Although this traditional role continues, the waning incidence of morbidity and mortality has reduced the annual admissions to this Service from 1,200 - 2,400 patients to less than 600 patients in recent years. As a result, this has permitted a reduction of the Infectious Disease Service from a 200 bed hospital to a single ward of 25 beds. Considering the intermittent outbreaks of various infectious diseases, of which poliomyelitis has been the most striking of recent times, it is unlikely that any further reduction can be effected. Indeed, the periodic provision of additional bed space to accommodate victims of future outbreaks and epidemics may be necessary.

The Respirator Service had its modest beginning in 1933 with the purchase of a single tank respirator. The limited demands on this service brought about by a relatively low incidence of respiratory paralysis in earlier poliomyelitis epidemics, coupled with a high mortality rate, limited its growth for many years to a total of four respirators. During the poliomyelitis epidemic of 1952 and 1953 the rapid expansion of this Service had to be provided in order to accommodate a peak load of 91 respirator patients. Not a single year has gone by since then that one or more patients with acute respiratory paralysis has not required admission to the Service. Besides undertaking to provide care and treatment for these acute cases and the chronic respirator patients left in the wake of the two epidemics, it is also required to accommodate respirator patients participating in the Home Care Program, as their need arises. Some aspects of this rewarding program are detailed elsewhere in this issue.

The Chronic Disease Service, which came into being with the opening of the Princess Elizabeth Hospital in 1950, is of the greatest current interest to the community. To meet the increasing need for this type of hospital accommodation the King Edward Memorial Hospital, following its retirement from the tuberculosis field in August, 1954, was extensively renovated to adapt it for the care of chronically ill. Accommodation vacated by the Infectious Disease Service has also been available to this Service. At the present time, 360 beds are available for patients suffering from chronic illness, and it is hoped to put another 40 beds into service by the end of the year.

The skilled nursing care and the dynamic therapeutic approach towards the care of the chronically ill patient, together with complete laboratory and diagnostic radiological facilities and the Department of Physical Medicine permit comprehensive diagnostic assessment and treatment service. Despite the diversion of an increasing number of beds for patients requiring maintenance rather than progressive care, the Chronic Disease Service has kept pace with the demands for accommodation. Our greatest value to the community lies in the proper utilization of the special services which the Winnipeg Municipal Hospitals have to offer in providing for the physical restoration and rehabilitation of the disabled patient. Although treatment facilities are available for patients of all age groups, the infirmities attendant upon advancing years account for the preponderance of geriatric patients admitted to the Chronic Disease Service. Indeed, these Hospitals have been privileged to occupy a unique position in our Province in the field of Geriatrics — providing an active and comprehensive program which is not elsewhere available.

A. J. W. ALCOCK, M.D.,
Medical Director,
Winnipeg Municipal Hospitals



The Winnipeg Municipal Hospitals

The City's Hospitals

E. F. Taylor, M.D.

Rising proudly above smooth lawns bordered with colorful flowers, two of the Winnipeg Municipal Hospitals give no hint, today, of their lowly origin or of their stormy passage from the vicinity of the Main Road and the Portage Trail to their present site, situated on a curve of the historic Red River at the foot of Morley Avenue.

The fight against infectious diseases in Winnipeg may be said to have commenced when a meeting was called December 18, 1872 to discuss ways and means for caring for the ill. Fiery and eloquent speeches were made by the leading men of the village of two hundred souls, among them the intrepid, red-headed Dr. Schultz. Four hundred dollars were quickly collected by contributions and, in January 1873, a bare, one-room, twelve bed ward over Dr. Schultz' store was ready to receive the sick. One man was employed to attend them. This was the beginning of the Winnipeg General Hospital. It was also the headspring of the Winnipeg Municipal Hospitals which, though opened for all types of diseases, catered largely to patients suffering from Typhoid or the Red River Fever, as it was commonly called. Many moves were made before the Winnipeg General Hospital came to rest on Bannatyne Avenue on March 13, 1884. Six beds were allotted for infectious diseases, separated from the rest of the rooms and reached by an outside staircase.

Space does not permit a detailed account of these early days, but one reads of smallpox victims removed from their homes in the dead of night to be conveyed past the tombstones of Brookside Cemetery to a pest house guarded by a barbed wire fence; typhoid and pulmonary tuberculosis crowded together; scarlet fever housed in a building on the old fair grounds on what is now Flora Place; of 911 typhoid cases admitted to the Winnipeg General Hospital in 1905 and others turned away; of the City Council hastily erecting a temporary annex and buying Dr. Beaths' old maternity hospital on Bannatyne for scarlet fever. One familiar cry is heard in all this turmoil, "Where can one find a nurse?"

The City Council in 1908 appointed a Committee of three men — Mr. Justice Mathers, Mr. G. R. Growe and Dr. J. R. Jones — to investigate the hospital accommodation in the city. Acting on their report, two by-laws were passed in 1910 for the erection of an isolation hospital and one for tuberculosis. As a result, on July 11, 1912, the Duke of Connaught, then Governor-General of Canada, accompanied by his lovely daughter, Princess Patricia, turned the Key to open the King Edward Memorial Hospital, then crossed the grounds to place the corner stone of the King George Hospital.

In the beginning, these hospitals were administered by R. T. Riley, Edward Brown, later to become the Honorable Edward Brown, and Alexander MacDonald. They chose for their medical staff Dr. A. B. Alexander as Medical Superintendent and the late Dr. A. T. Mathers, then a recent graduate, as Assistant Medical Superintendent. Dr. Alexander was noted for his diplomacy in meeting the public, Dr. Mathers for his scientific and organizing ability. Among the consultants were such well known names as Drs. B. J. Brandson, J. D. McEachern, Gordon Fahrni, E. J. Washington and F. D. McKenty.

The King Edward, with a capacity of over a hundred beds, had been built for patients with advanced tuberculosis and for many years those discharged as arrested cases were pitifully few. It was in the Out-Door Department that the best work was done. Hundreds of contacts were investigated each year. Not, however, until 1947 did the gloomy cloud which hovered over these patients tend to lift. Anti tuberculosis chemotherapy changed the picture so rapidly that the King Edward was closed as a Tuberculosis Hospital in 1954 to reopen for the care of geriatric cases and chronic disease in 1956.

Epidemic after epidemic has passed through the King George. Due to preventive measures and the antibiotics, smallpox, diphtheria and poliomyelitis, to mention a few, have come under control. Where once the admissions amounted to thousands in a year, they can now be counted by the hundreds. The King George is slowly being transformed for patients requiring extended periods of care and for rehabilitation, but with the wards always equipped to meet a new or unexpected epidemic.

Before one patient had entered the Princess Elizabeth, the building was flooded in 1950, the spotless rooms sullied with mud. It had been designed for those who were chronically and, perhaps, hopelessly ill. But the idea that the patients entering there would require little more than nursing care was quickly abandoned. The medical staff who were experienced in the care of tuberculous patients and those paralyzed from poliomyelitis and who were also keenly interested in the opening field of geriatrics thought that much could be done in rehabilitation to rescue the patients of this type from a life of inactivity and despair, and to provide one of hope and moderate enjoyment. The City's Hospitals, in the past few years, have assumed increasingly the function of a centre in the Community where such rehabilitative facilities and services are available.

This is a short history of the City's Hospitals. They have weathered the storms of the past and stand strong to meet the future.

Geriatric Rehabilitation

M. H. L. Desmarais
L.R.C.P., M.R.C.S. (Eng.), D. Phys. Med. (Lond.)
Director, Department of Physical Medicine & Rehab.
Winnipeg Municipal Hospital

Introduction

The population in the 60 and over age group is increasing significantly. It is amongst this age group that the greatest incidence of prolonged illness occurs[1]. However, it has been demonstrated[2, 3] that a positive attitude towards treatment utilizing a combination of medical, surgical and nursing skills and the measures provided by physical medicine, can reduce significantly the need for long term hospital accommodation (chronic beds) of an appreciable number of these patients.

It is the purpose of this paper to outline the growth and development of the Department of Physical Medicine at the Winnipeg Municipal Hospitals; to describe some of its facilities; and to indicate the scope of its work, some of the methods used and some of the results achieved.

Rehabilitation Facilities

Development

At the Winnipeg Municipal Hospitals, the facilities and organization for Geriatric Rehabilitation had their inception with the opening of 208 beds at the Princess Elizabeth Hospital in 1950. The Department of Physical Medicine at that time had a staff complement of two Physiotherapists, a space allotment of about 800 square feet and a variety of the usual tools of the trade which had been considerably amplified by a donation, in sympathy for the losses of the Red River flood of 1950, from the Council of Round Tables of Great Britain. The poliomyelitis epidemics of 1952 and 1953 imposed increased demands for rehabilitative services. In consequence an expansion in the Department of Physical Medicine resulted, entailing the appointment of a qualified Physiatrist as director, additional Physiotherapists, Occupational therapists, various technicians and aides. Space allotment was increased until, with the opening of the Hydrotherapy Unit in 1957, a grand total of some 7800 square feet is now provided for the operation of the Department. In addition, a ward of 20 beds for special rehabilitation problems has been provided. During 1955, the King Edward Hospital was converted to provide 122 Geriatric and Chronic Disease beds. The Department of Physical Medicine has now developed to the stage where full scale and concerted Geriatric Rehabilitation can be accomplished.

Present Facilities

Some indication of the physical facilities of the Department has already been given. Whirlpool baths, parallel bars and mirrors for walking re-education; slings, weights and pulleys; all of these and all the other expected paraphernalia of a Department of Physical Medicine are, of course, provided. In addition, however, there are other facilities, a little out of the ordinary perhaps, but designed and developed to provide for special needs in this field of rehabilitation.

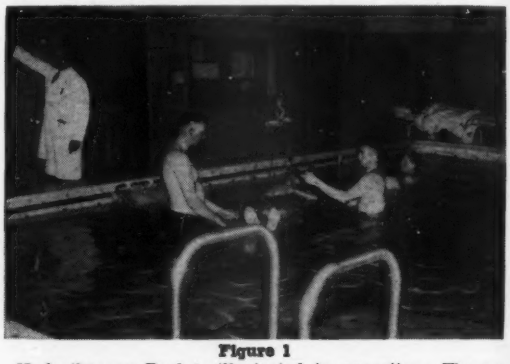


Figure 1

Hydrotherapy Pool is illustrated in operation. The remainder of the Hydrotherapy Unit comprises a Hubbard Tank, Whirlpool baths, as well as showers, toilets, dressing rooms and a large waiting room.

The fine Hydrotherapy Unit (Figure 1) provided through the public spirited efforts of the Kiwanis Club of Fort Garry, with financial assistance from the City of Winnipeg and Provincial and Federal Governments, is one of the first of its kind in Western Canada and has been in operation since October 1957. The value of such a Unit in providing therapy for the victims of poliomyelitis is well recognized. In Geriatric Rehabilitation it has even wider application. At the appropriate stage, the patient with the fractured hip can walk and exercise in the pool as an adjunct to his other program, the buoyancy of the water minimizing or obviating the effect of weight bearing on his yet ununited fracture. The hemiplegic finds his paretic limb easier to move and gains confidence. The arthritic, too, experiences facility of limb movement, that relief of joint pain which accompanies the warmth of the water and the progressive exercise which leads to better muscle tone and power resulting in increased mobility. The patient with vertebral bodies collapsed from senile osteoporosis, is allowed to redevelop muscle activity and tone with whatever beneficial effect this may have on the bones, but without the addition of weight stress on those bones since the water provides buoyancy. A total of some 3,096 treatments in the Hydrotherapy Unit have been provided in the past year.

The Device Workshop, fully equipped with hand and power tools and bench space, has a twofold purpose. Firstly, a supervised work area is provided for Occupational therapy where the testing of patients' work capacity and the development of their abilities and tolerance can be carried out.

Secondly, a large variety of devices—tripod canes, simple "walkers," long angle-handed combs and brushes, special eating utensils—each developed for an individual patient disability requirement, are provided. Repairs and modifications of appliances and equipment are also carried out here.

In the Splint room, a large number of light weight but durable plastic reinforced plaster "rest splints" are produced. These are essential in the prevention of disabling deformities, particularly in the hemiplegics and arthritics. Another first in this part of Western Canada, the "Long John Silver,"—a plaster-socket pylon[4]—is fabricated here.

Staff

Available personnel seldom fulfill the allowable staff complement of eleven Physiotherapists, two Occupational Therapists, two Remedial Gymnasts, two Technicians to service the Device Workshop and the Activities of Daily Living program respectively, and assorted aides, receptionists, clerks and secretaries. Personnel of equal importance in a successful Geriatric Rehabilitation program, however, are not those formally included in the Department of Physical Medicine complement. Rather, they are the nursing staffs of the wards of the Winnipeg Municipal Hospitals—the Princess Elizabeth, the King George and the King Edward. The registered nurses, the practical nurses, the orderlies and the ward aides who comprise each ward staff, are an integral part of a functioning Geriatric Rehabilitation program. Getting out of bed, feeding one's self, dressing one's self—each of these is an exercise in physio and occupational therapy. It must be remembered that any Department of Physical Medicine, no matter how completely staffed, can provide active treatment for but a limited portion of available treatment time in the patient's day. On the other hand, the ward staff has at its disposal, a large proportion of the available treatment time, which if logically used, can reduce the total rehabilitation period significantly. In cognizance of this role in the over-all approach, the Director of the Department and each of its members take every opportunity to encourage and to instruct each ward staff in the measures which will achieve the goal of rehabilitation—the independence of the patient.

Case Material — 1959

Case Referrals

Patients are referred to the Department of Physical Medicine for consultation rather than "for physio." Consequently each case is seen and assessed by the Director of the Department. It is felt that only with a sound appreciation of the physical, mental and social aspects of an individual case can intelligent rehabilitation measures be programmed. Hence an attempt is made to individualize each course of treatment. This effort entails the regular discussion of the patient's progress at ward rounds or review clinics with the physician in charge of the case, the nurse, the social worker and the members of the Department concerned with that case. Only by such co-operative and co-ordinated understanding of all aspects of the individual patient's problems can satisfactory rehabilitation be achieved.

Table 1 indicates the disease and age characteristics, the results of treatment and the disposal of the 242 patients, 60 and over, whose course of treatment in the Department was initiated and completed during 1959.

Table 1

Diagnosis	No. of Patients	Age Groups in Years 60-69	70-79	80+
C. V. A.	81	30	35	16
Fractured Hips	56	9	20	27
Other Fractures	13	1	8	4
Amputees	9	4	3	2
Others	83	27	35	21
Total	242	71	101	70

Cerebrovascular Accidents

It is evident from Table 1 that cerebrovascular accident patients constituted the largest single group of referrals during 1959. Unfortunately, many of these individuals are admitted to the Winnipeg Municipal Hospitals relatively late in the course of their disease. Already they are "problem cases," and much valuable time in a rehabilitative program has been lost. The initial assessment in this group particularly, provides a guide as to program and prognosis. On the one hand, with intact sensation and some voluntary movement of the hemiplegic limbs, the goal of independent ambulation can be set. In such a case, walking rehabilitation on the ward, in the gym and the hydrotherapy pool; bowel and bladder training; practise and encouragement to get in and out of bed, to eat and dress independently; joint mobility exercises and rest splinting to prevent contraction deformities; stair climbing; and later week-end home leaves form the basis of the progressive return to normal living. On the other hand however, sensory defects such as hemianopsia, loss of body image or reduced proprioceptive appreciation of the hemiplegic side provide a more complicated problem. In this case, independent ambulation is unlikely and the primary objective is set at wheelchair ambulation. Here, instead of upon walking re-education, the emphasis is laid upon training in getting in and out of the wheelchair under all possible circumstances.

Fractured Hips

The second major group of patients referred were those with pinned fractured hips. Early treatment here, consisting of sling and bed exercises, is designed to maintain muscle tone and joint mobility for the later stage of training in non-weight bearing crutch walking. As mentioned previously, the hydrotherapy pool is valuable at this later stage. Once the skill of crutch walking has been mastered, the patient can be discharged to a home environment and reviewed periodically as an Out Patient. Often, however, social circumstances are unsuitable and the program must be continued in hospital until firm bony union permits weight bearing.

Amputees

The leg amputations form a small but interesting group. A progressive course of physiotherapy begins immediately after the operation with proper limb positioning in bed to prevent flexion adduction hip deformities, and flexion deformities of the knees. As soon as sutures are removed, the stump conditioning measures of bandaging and "pounding" are begun. Elastic crepe bandages are applied in a special manner to produce a conically shaped stump. Pounding or banging the stump with small sand bags toughens the skin and reduces the incidence of phantom limb pain and tender neuromas. With these measures, and muscle strengthening exercises, the patient is soon ready for walking. Amongst the elderly, however, are some who will be unable to use an artificial limb. Consequently, the "Long John Silver" has been devised[4] for a trial period to determine suitability for the expensive permanent prosthesis. A plaster of Paris socket is fashioned directly on the stump. Metal strips are embedded in the plaster. After hardening, a stout oak peg is secured by the metal strips and the "Long John Silver" is ready for use. (Figures 2 and 3).

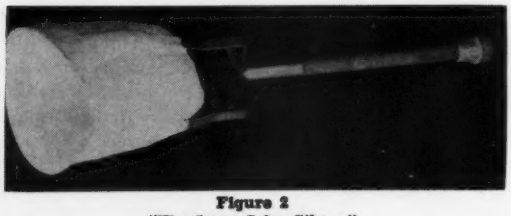


Figure 2
"The Long John Silver."

Results

Table 2 summarizes the results obtained in the 242 patients. Improvement was obtained in the patient's status in 58% of the cases. The duration of treatment was also interesting. Of the "improved" cases, two-thirds had achieved that status by the end of three months of treatment; the remaining one-third received between three and six months of treatment.

Table 2

Diagnosis	Results of Treatment: Improved	Not Improved	Died	Total
C. V. A.	43	27	11	81
Fractured Hips	32	18	6	56
Others	67	31	7	105

The disposal of the patient group is shown in Table 3; 60% of the patients were able to return home; 20% were transferred to other institutions such as Nursing Homes as no longer requiring hospital care; and 10% remained as hospital patients.

Table 3

	Died	Home	Remaining In Hosp.	Transferred To Other Inst.	Total
C. V. A.	11	49	8	13	81
Fractured Hips	6	31	3	16	56
Others	7	70	13	15	105
Total	24	150	24	44	242

Figure 3
The "Long John Silver" in use in the case of an above-knee amputee.
The pylon is secured by a harness which goes over the shoulder opposite to the amputation.

Another result of such a program of Geriatric Rehabilitation is the effect upon ward staff and medical staff, of seeing patients previously considered to be "vegetables"—bed ridden, incontinent and apathetic—given the opportunity of regaining some degree of independence and of returning home. This effect cannot be tabulated.

Discussion

Recumbency engenders geriatric disability[5]. The aim of Geriatric Rehabilitation is to provide the means whereby, early in his illness, the elderly patient can regain an independence in his existence consistent with the degree of his disability. It has been our experience that there is a reciprocal relationship between those parameters—as the degree of independence waxes, the degree of disability wanes. The success of a program of Geriatric Rehabilitation is measured by the numbers of patients who can be returned to a home environment. A figure of 60% is perhaps not outstanding, but it lends emphasis to the feeling that it could be considerably better had the cases reached the Municipal Hospitals sooner—soon after consciousness is regained for the cerebrovascular accident, as soon as sutures have been removed from the pinned fractured hip. The effect upon the community at large merits mention. In achieving the

discharge of 150 patients, active Geriatric Rehabilitation measures have spared the community the expense of providing and maintaining 150 chronic care beds.

Summary

The growth of the Department of Physical Medicine at the Winnipeg Municipal Hospitals has been described, together with some of the treatment facilities which it has developed.

The numbers of patients treated in the Department during 1959 have been indicated. The necessity of an adequate initial case assessment and of continued supervision and re-assessment in any outlined course of therapy has ben emphasized.

The results of treatment in 242 patients, as well as their ultimate disposal have been tabulated.

I wish to thank Drs. J. A. MacDonell and J. Hildes for their very active co-operation in writing this paper. I am also indebted to Maurice I Law, Chief Physiotherapist, for his assistance in compiling the statistics for Tables 1, 2 and 3.

Bibliography

1. Commission on Chronic Illness: Care of the Long Term Patient. Harvard University Press, Cambridge, 1956.
2. Cosin, L.: A Statistical Analysis of Geriatric Care. Proc. Roy. Soc. Med., 41: 333-336, 1948.
3. Warren, M. W.: Care of the Chronic Aged Sick, Lancet, 250: 841-3, 1946.
4. Gillis, L.: Artificial Limbs, p. 67, Pitman, 1957.
5. Annotation: Bed Rest and Rest Eternal, Lancet, 264: 181, 1953.

♦ ♦ ♦

The Role of a Home Care Program in the Management of Chronic Disease

J. A. Hildes and R. G. Handford

Introduction

A current medical problem of some importance is the increasing requirement for long term care of patients with chronic diseases, many of whom are in the geriatric age group. One of the alternatives to long term or indefinite stay in hospital is the adequate provision in the home for care of selected patients who would otherwise occupy hospital beds, either continually or repeatedly. Not only may the medical needs be met in this way, but patients and their families, given adequate facilities and support, often prefer care in the home. There are economic advantages also to the provision of home care for selected patients. The cost of care is less than the cost in hospital and, also, by this means a saving in hospital beds is achieved which reflects back on the availability of beds in the acute hospitals.

Arrangements for home care were first made at the Winnipeg Municipal Hospitals to look after the large number of chronic respirator cases resulting from the poliomyelitis epidemics in 1952 and 1953, many of whom expressed a strong desire to be cared for at home. The purpose of the present communication is to indicate the results of this program after five years of operation and to indicate its extension to include other chronic diseases.

Home Care for Respirator Patients

The considerations leading to the inauguration of the home care program for respirator patients, the factors involved in case selection, the needs of these patients regarding medical care, equipment and social services, as well as the arrangements for continuing surveillance, have been previously reported along with preliminary results[1].

Patients were only discharged if the additional risks of being away from the hospital were considered small. To ensure this, the program was based in the hospital which undertook to maintain regular surveillance of all aspects of the case. This included, as well as a regular medical follow-up, the maintenance of special equipment and the arrangements for such additional needs as attendant or house-keeper service and structural alterations to the home to accommodate equipment and to facilitate patient care. The cost of these extra services was shared by the Provincial Department of Health and the Society for Crippled Children and Adults.

The involvement of several agencies in the care of one patient necessitated the development of experience which was achieved by case conferences to which the family physician and representatives of the agencies were invited. As experience matured, the routine surveillance was handled more and more by one or two hospital physicians who could call in the services of the other agencies as required.

Results of Home Care for Respirator Patients

At the time of the previous report on this program[1], five patients had been discharged home who required the use of a respirator regularly for 8-12 hours each day. In addition, nine patients who no longer required regular artificial ventilation but who were severely disabled by a limited respiratory reserve, were also discharged under these auspices.

The situation at the end of 1959 is shown in Table No. 1. Fifty-one patients had been discharged to home care. There were approximately an equal

Table 1

Results of Home Care Program for Respirator Patients 1955 - 1959

Total Number of Patients Discharged to Home Care		51*
Discharged with Respirators	27	
Ex-Respirator Cases	17	
Others	7	
Respirator Patients Still in Hospital		31
Being Prepared for Home Care	5	
In Hospital for Medical Reasons	14	
In Hospital for Social Reasons	12	

*Including 5 deaths.

number of males and females in the young adult age group. There were only six under 20 years of age and three over 50. More than half of them continued to use artificial ventilation for regular periods each day. The others had either achieved a tenuous freedom from their respirators or had never regularly required artificial ventilation, although their respiratory function was seriously limited by paralysis and further compromised by chest deformity or lung changes. In all of these patients, minor illness, particularly respiratory tract infections, may prompt readmission to hospital.

Approximately half of the thirty-one respiratory cases still remaining in hospital are so disabled that home care is considered too hazardous to risk. Most of these have extreme trunk and limb paralysis and they have never learned to "frog breathe"[2]. The remainder are potential candidates for home care, and arrangements are being made to effect the discharge of five of them to their homes. The remaining twelve cases could have their medical needs met at home as easily as those already discharged, but their social circumstances preclude discharge. To some extent, this is due to the delay in setting up the home care program for two or three years after their admission to hospital in 1952 or 1953. If both the patients and their families had anticipated discharge from hospital from the beginning, some of the present difficulties would not have arisen.

Equipment and Services

The various items of equipment supplied for home care were determined by the needs of the patient. Twenty-seven required some form of regular assisted ventilation and ten of these, mainly in the rural districts, were given two appliances in the event of mechanical or electrical failure and to maintain ventilation during transportation to hospital in the event of a respiratory infection. Twenty of twenty-seven preferred to use a rocking bed for regular use; others used tank respirators or chest respirators regularly.

The patients are encouraged to be up and as active as possible within the limits imposed on them by respiratory or trunk and limb paralysis. A few can walk but most of them use wheelchairs. Hydraulic lifts, commode chairs on wheels, ramps or lifts for stairs or curbs and a variety of self-help devices for feeding, reading, dressing, telephoning, all ease the physical work required of the attendant.

Most of the home care patients have a part-time or a full-time paid attendant to help with their personal needs and, depending on the circumstances, to sometimes assume responsibility for housework and the care of children.

Morbidity and Mortality

Table 2 shows the number of cases requiring re-admission to hospital and the number of deaths of home care patients. Thirty-seven per cent of the patients have had no re-admissions after discharge, including six of the twenty-seven requiring regular artificial respiration. An equal number have had 1 - 2 re-admissions and 18% have had four or more re-admissions. Approximately 70% of the re-admissions were for acute respiratory infections. The next commonest medical indication was urinary tract infections, usually associated with calculi[3]. Some of the re-admissions were for further training in the use of self-help devices and a few patients living far removed from the respiratory centre have been re-admitted for one or two days for regular review.

Table 2
Morbidity and Mortality

	Respirator	Ex-Respirator	Non-Respirator
No re-admissions	6	8	5
1 - 3 re-admissions	12	5	1
4 or more re-admissions	6	2	1
Deaths	3	2	0
Total	27	17	7

Three of the five deaths among the home care patients were sudden and unexpected. One of these was examined at post mortem, but no immediate cause of death was found. One other died at home with acute respiratory infection. The remaining death was due to chronic pyelonephritis associated with renal calculi. This patient had recurrent admissions for renal failure and hypertension.

Level of Activity of Home Care Cases

Although many of the home care patients require either a part-time or a full-time attendant to assist the family, and all have additional equipment such as wheelchairs, hydraulic lifts, special ramps and self-help devices, they are by no means all helpless. Approximately 20% are either breadwinners, attending school or doing their own housework. The majority have some form of useful activity with the aid of a wheelchair, and self-help devices, and only about 20% are incapable of helping themselves physically.

Home Care for Other Chronic Diseases

The success of the home care program for respirator patients was one of the factors which encouraged the establishment of home care services for patients with chronic diseases in the Winnipeg General Hospital[4]. With the development of a program at the Winnipeg General Hospital it was considered unnecessary to duplicate all the special services required, particularly a general medical out-patient department and medical and social service staff to make home visits. Arrangements have been made, therefore, to integrate the home care of patients with chronic diseases other than polio at the Winnipeg Municipal Hospitals with the plan set up in the larger general hospital. These arrangements have been only recently completed, so that at the present time only a relatively small number of such patients have been discharged. The co-ordination between the chronic disease service of the Municipal Hospitals and the out-patient department of the Winnipeg General Hospital is a natural one, since many of the patients admitted to the chronic disease wards of the Municipal Hospitals are referred from the Winnipeg Genera'

Hospital, and on discharge are referred back to the out-patient department of that institution.

Patients on the chronic disease service who are considered to be candidates for home care are seen by a medical member of the home care team at the Winnipeg General Hospital who then arranges for the necessary preliminary investigation of the home, for home medical and nursing visits, and for the provision of drugs and supplies from the out-patient department of the General Hospital.

Results

Under these arrangements ten patients have been discharged. Three suffer from cerebral arteriosclerosis with hemiplegia; three have chronic disabling arthritis; two have multiple sclerosis and there were three cases of paraplegia, one of whom subsequently died of pyelonephritis.

As more experience is gained in this relatively new venture, it is anticipated that more patients will be discharged home. One other case is now under active consideration.

Discussion and Conclusions

This brief review of what has been accomplished by home care at the Winnipeg Municipal Hospitals does not cover all the details which can only be brought out by a full consideration of individual cases, particularly as many of the arrangements must be moulded by local conditions and existing facilities. We have attempted to show that, in the home, not only medical needs can be satisfied, but also the dignity and happiness of the patients and their families may be better preserved. These two advantages, in addition to the economic saving from such a program, are clear indications of the need of home care. To date, more than fifty patients are maintained out of hospital, and, although it is hoped that polio will be a diminishing disease, there is no dearth of patients with other chronic diseases who would benefit from home care.

References

1. Desmarais, M. H. L., Alcock, A. J. W. and Hildes, J. A.: Home Care Program for Respirator Patients. Canad. M.A.J. 75: 654, 1956.
2. Collier, C. R., Dail, C. W. and Affeldt, J. E.: Mechanics of Glossopharyngeal Breathing. J. Appl. Physiol. 8: 580, 1956.
3. Taylor, J. R., Alcock, A. J. W. and Hildes, J. A.: Urinary Tract Calculi in Poliomyelitis. Canad. M.A.J. 75: 29, 1956.
4. Fyles, T. W., Gemmell, J. P., Hildes, J. A. and Handford, R. G.: Report on Home Care Program, Out-Patient Department, Winnipeg General Hospital—1959. In preparation for press.

♦ ♦ ♦

Co-operative Attack on Arthritis

E. J. Thomas, M.D.

Introduction

One of the major problems of the medical profession has been the care of the chronically ill. Throughout medical history, groups of chronic diseases have been neglected because of the difficulty in maintaining the interest of medical personnel. The profession has focused its attention on diseases that can kill or be cured, while diseases that merely cripple have been overlooked. When care has been provided, it has been difficult to maintain high standards. Hospital beds have not been available for long term treatment. These problems, added to the economic loss suffered by the chronically ill due to their inability to carry on with their normal activities, have presented a complex picture.

Development of Facilities

Nationally

In Canada, prior to 1949, very little had been done to organize diagnostic or treatment facilities for patients suffering from arthritis and rheumatic diseases. In that year, representatives of health departments, universities, and the medical profession met in Ottawa to form the Canadian Arthritis and Rheumatism Society. Within the next few years divisions were formed in eight provinces to provide a concerted attack on these diseases which cripple 115,000 Canadians and cause a loss of 9,000,000 work days per year at a cost of $75,000,000.00.

The objectives of this Society are:

1. To promote a program of research and professional education in the rheumatic diseases; to discover their causes; develop methods of prevention and to extend knowledge about their treatment.
2. To promote the establishment and expansion of facilities for the diagnosis and treatment, rehabilitation and welfare of sufferers.
3. To provide sufferers and the general public with factual information about early recognition of symptoms and the need for prompt treatment.
4. To raise funds for these purposes.

In Manitoba

In 1950 the Manitoba Division was formed under the guidance of Dr. J. D. Adamson who became the Society's first president. Arthritis Clinics were established as part of the outdoor departments of the Winnipeg General and the St. Boniface Hospitals. The services of Internists with a special interest in rheumatology were obtained. Due to the lack of physiotherapy service available in Winnipeg, a physiotherapist was employed by the Society in 1951. She operated a mobile unit providing a home-care service.

By 1955 this service had been extended, and weekly Physiotherapy Clinics were being held in seven centres outside Winnipeg and a branch had been formed in Brandon. In that year the Society established its office and treatment centre in the remodeled King Edward Hospital, recently opened for the treatment of chronic diseases.

In 1957 rural consulting clinics were instituted under a Federal Health Grant. These were designed not only to assist local physicians with problems in diagnosis and treatment, but also to stimulate interest in the arthritic and allied diseases. Recently these have been extended, and it is hoped that in the near future all larger centres throughout the Province will be visited at regular intervals, not only by a Rheumatologist, but also by Physio- and Occupational Therapists. These have proven to be a rewarding case-finding mechanism.

Winnipeg Treatment Centre

For many years the attending medical staff of the Winnipeg Municipal Hospitals had shown great interest in the treatment of arthritics and had encouraged their admission to the chronic disease wards. The advent of government sponsored hospitalization insurance made possible the creation of a much more extensive arthritic unit for the in-patient care of those requiring prolonged or intensive and specialized treatment.

In September of 1959 the Winnipeg Municipal Hospitals and the Canadian Arthritis and Rheumatism Society initiated a co-operative attack on arthritis. Beds in the King Edward Hospital were set aside for suitable cases and the staff of the two organizations worked out a plan whereby patients would receive the maximum benefit of all available treatment facilities.

Weekly arthritis conferences were instituted at which patients are assessed on admission and at regular intervals during their hospital stay. Problem cases being treated as out-patients are also presented. These conferences are attended by the attending medical staff, house physicians, the hospital physiatrist, consulting rheumatologists, the hospital social worker and physiotherapists and occupational therapists from both organizations. As well as medical and nursing care the following facilities are available and are utilized as indicated: physiotherapy, hydrotherapy and occupational therapy. Trained personnel are available to make rest plasters, splints, self-help devices and to instruct patients in activities of daily living. Full use is made of the vocational and rehabilitation services offered by other health agencies.

Case Material

Patients

From January 1958 until December 1959, thirty-seven cases of rheumatoid arthritis were admitted to the staff wards of the Winnipeg Municipal Hospitals. Of these, ten were male and twenty-seven were female. The age range was eighteen to eighty-three years, with an average age of 56.1 years. These thirty-seven patients were classified on admission according to the standard "Functional Capacity Code" which is as follows:

Functional Capacity Code

1. Unimpaired or slightly impaired

Functional capacity adequate to perform all normal activities of daily living without the use of prosthetic or other appliances, and despite slight intermittent discomfort or limited mobility of one or more joints.

2. Complete self-care

Functional capacity adequate to perform the majority of normal activities of daily living with or without the use of prosthetic or other appliances; able to feed, dress, use toilet facilities and leave house without assistance from a second person.

3. Partial self-care

Functional capacity adequate to perform the majority of normal activities of daily living with or without the use of prosthetic or other appliances; able to feed, dress and use toilet facilities; may require occasional assistance from a second person; unable to prepare meals or unable to leave house without assistance from a second person.

4. Severely impaired

Functional capacity insufficient to perform the majority of normal activities of daily living, with or without the use of prosthetic or other appliances; require regular assistance from a second person in performing one or more of the following activities: feeding, dressing, using toilet facilities, or getting about indoors in wheel-chair or otherwise.

On admission, eight Stage 4 cases were considered unsuitable for any physical or remedial treatment but required nursing care only. The average duration of the disease in this group was thirty-two years (with a range of nineteen to sixty-one years). Two of these patients died; one from advanced rheumatoid arthritis and secondary amyloidosis confirmed at autopsy; and the second from advanced rheumatoid arthritis, bronchopneumonia and an astrocytoma of the right occipital lobe. This later lesion was not suspected during life but was found at post mortem.

Results of Treatment

The twenty-nine patients who were considered suitable for treatment had had their disease for an average of 13.5 years (range 6 months to 57 years). These were classified on admission as follows:

Stage 1	3
Stage 2	6
Stage 3	9
Stage 4	11

The average length of hospital treatment of this group was 11.5 weeks. On discharge the twenty-nine cases were classified as follows:

Stage 1	7
Stage 2	10
Stage 3	9
Stage 4	3

(See Tables 1 and 2 and Histogram 1)

Table 1

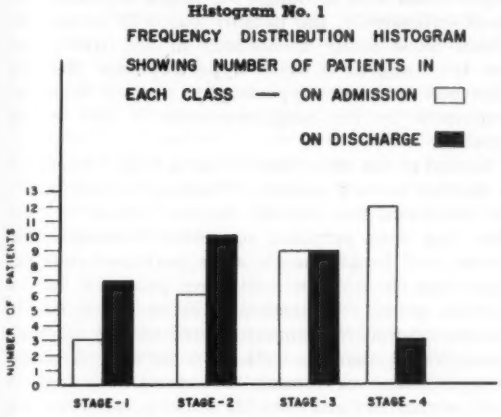
	Improved	Not Improved	Rejected for Treatment	Died
All Patients	20	9	8	8
Excluding Stage 4	12	6	0	0
Stage 4	8	3	8	2

Table 2

Stage	1	2	3	4	Totals
Number Admitted	3	6	9	11	29
Number Discharged	7	10	9	3	29

X^2 analysis [2 d.f.] p less than .001 (highly significant) (Because of small expected numbers, two cells were combined giving two degrees of freedom).

Histogram No. 1



Discussion

It is fully appreciated that this is a small series and has been analysed without a control group. We have not attempted to analyse or define the results as to treatment effects. Treatment consisted of medication as indicated and physical therapy in its various forms, also as indicated. We feel justified in concluding from these results that the earlier treatment is started in rheumatoid arthritis requiring hospitalization, the higher the percentage of improved on discharge. (See Histogram 1).

Of a total of 37 patients admitted, 20 were discharged improved—54.05%. This group includes all of Stage 4 patients, eight of whom were unsuitable for treatment. If these eight cases are excluded, 20 out of 29 patients were discharged improved—69%.

It is our hope that as the service becomes known we will get cases earlier for intensive therapy, for at the present time this appears to be the best method of preventing permanent incapacity from multiple deformities. Patients whose disease remains active often require several periods of hospitalization.

The number of Stage 3 and Stage 4 cases that did improve on this regime is worthy of note.

Summary

1. The development of arthritic services in Manitoba since 1951 has been briefly outlined.

2. The present arthritic treatment centre at the Winnipeg Municipal Hospital has been described.

3. The results of treatment of thirty-seven cases of rheumatoid arthritis treated in the Winnipeg Municipal Hospitals in 1958-59 have been analysed.

♦ ♦ ♦

Chronic Sick

The field of the chronic sick is one in which very marked changes have taken place with the introduction of the National Health Service. There is no doubt that in the past hospitals for the chronic sick have tended to be institutions providing rest in bed for this group of patients, but little else. Indeed, in the many reviews of patients in these long-stay hospitals which took place when Regional Hospital Boards were formed it was clear that many patients were suffering from contractures and other disabilities from prolonged rest in bed, and that the idea of getting these patients up, and the prospect of their discharge, was one unwelcome to patients, relatives and staff. The policy was one of custodial care. With this outlook there could be no solution to the problem of the chronic sick than an increased provision of beds, an increase which would need to take place step by step with the increasing age of the population.

"If we approach the problem of 'disposing' of the elderly infirm it will never be solved because of their increasing numbers. The problem of re-settling and finding continued care for the elderly is, I feel, capable of solution. . .

"The feeling of despair in the medical profession when faced with a problem they feel insoluble except on the lines of more and more overcrowded beds for elderly "chronics" must be replaced by a reasoned optimism of the possibilities of rehabilitation of the elderly, as a result of which it may well be that the supposed deficit in hospital accommodation is minimal or non-existent."

L. Z. Cosin as quoted in report of Ad Hoc Committee on Trends in Medicine to British Ministry of Health, 1956

Anabolizers

Walter Zingg, M.D., M.Sc., F.R.C.S. (C)

Anabolism is " a process by which a cell takes from the blood the substance required for repair and growth, building it into a cytoplasm, thus converting a non-living material into the living cytoplasm of the cell"[1]. Substances which induce, increase or accelerate these reactions have been called anabolic hormones, anabolic steroids or anabolizers. Although the last term is an ugly linguistic creation it may be the best one, because it is non-committal. In this paper the development of the substances and its actions will be reviewed and the clinical applications will be discussed.

A. Introduction

Before delving into the problem further it may be necessary to point out that anabolism is a complicated process depending, among other things, on various endocrine organs. It is also well known that the stimulatory effect of some hormones is restricted to certain organs or tissues. The specific effect of the sex hormones on sexual organs and the overgrowth of the "acra" in the presence of an excess of growth hormone may serve as examples. Consequently the assessment of anabolic substances presents a number of problems, since the general anabolic effects have to be differentiated from the specific effects, for instance a gain in weight could be produced by an overgrowth of sexual organs. In the absence of balance studies the effect is usually evaluated by following the change in the patient's weight and by such uncertain entities as increase in appetite or improved well-being. A weight gain, of course, may be due to water retention and the patient's appetite is influenced by many factors. In certain patients a placebo given with a convincing suggestion, "this pill is going to improve your appetite," may have an effect quite similar to an anabolic substance. Even with balance studies it may not be possible to decide whether the primary action is on the appetite, followed by an increased intake of food, or on the metabolism, followed by an increased appetite. Since we know only vaguely what stimulates the appetite, this question cannot be answered. As will be mentioned later, the development of methods for the quantitative estimation of the effect of anabolic substances in experimental animals has enabled the pharmacologists to assess and to compare these various compounds.

B. Hormonal Control of Anabolism

Although some animal studies on the anabolic effect of androgens had been made previously[2], it was not until 1943 that important observations in men were made. At that time two papers were published, which, although they could not explain the phenomena, are masterpieces of description and clarity of observation. Pierre Masson described two cases with testicular tumors originating from the Leydig cells[3]. One had been observed for many years and was known to have a high concentration of androgens in the urine. At autopsy the pathologist noted that, although the tumor had metastasized extensively, the patient was well nourished. There were many metastases in the liver, and the liver appeared to be hypertrophied. He also observed that the hypertrophy seemed to occur especially in the neighbourhood of the tumor masses.

Almost at the same time Fuller Albright delivered a Harvey lecture entitled "Cushing's syndrome"[4]. He discussed the various theories which at that time had been proposed regarding Cushing's syndrome and he also made some pertinent remarks regarding the anabolic substance produced by the adrenal glands "N-Hormone," as he called it. He postulated that N-hormone is produced by the normal adrenal gland as well as by tumors. Albright observed that in animals testosterone causes not only sexual but also somatic development, and his balance studies showed an increased positive nitrogen balance. The fact that calcium and phosphorus were retained in the same proportion as they are present in protoplasm suggested the new formation of tissue. Albright also briefly discusses the alarm reaction and he mentions a problem which has remained controversial to this date. During the alarm reaction catabolic hormones are released, and, if this is thought to be beneficial, it should not be interfered with. Accordingly, in Albright's opinion testosterone is not indicated during the acute stage following injury. The stimulus for tissue repair already is maximal at the site of injury.

Growth Hormone

An overproduction of growth hormone by the anterior lobe of the pituitary gland leads to acromegaly or gigantism. Although growth hormone meets the requirements of an anabolizer as described above, it does not have practical applications and it will not be included in this review. It may be useful in certain conditions, such as following extensive burns, but at the present time the use of growth hormone is still in the experimental stage[5, 6, 7, 8, 9].

Testosterone

A general stimulating effect of sex hormones has been known for a long time. In boys an overproduction of androgenic hormones is followed by precocious puberty, hirsutism and excessive growth of the musculature — the "Infant Hercules Syndrome" of the older writers. It was also known that in female pseudo-hermaphrodism a heavy musculature usually is present. The effect of these hormones therefore is not restricted to the sexual organs; they also have what later was termed an anabolic effect. Less dramatically the same effects can be observed during the normal development of

boys and girls during puberty. In addition to these facts a general effect on well-being and tissue regeneration was postulated.

The therapeutic use of testosterone in hypogonadal males is not discussed in this paper. In normal males the reaction is similar, although quantitatively different[10, 11, 12, 13]. The administration of testosterone to individuals in clinical endocrine balance is followed by nitrogen retention. A study conducted at the Mayo Clinic on patients with spinal cord injuries[14] showed that testosterone administration maintained a better nutritional status. Compared to a control group the treated patients showed less hypoproteinemia, a lower incidence of decubitus ulcers and a lower urinary excretion of nitrogen and creatine.

Testosterone, of course, influences the sexual organs and this limits its clinical usefulness as an anabolic agent. The studies cited above showed that the sexual and the anabolic effects really are two different things, and, accordingly, the possibility exists that a substance may be found which, although being anabolic, exerts little or no effect on the primary and secondary sex characteristics. It was already known that by adding a Methyl-group to the formula of testosterone a substance could be produced (Methyltestosterone) which clinically was similar to, but not identical with testosterone. Accordingly, the search for such a substance began and is still continuing today.

Experimental Evaluation

For the testing of testosterone derivatives a method had to be developed which allowed the differentiation of the anabolic from the androgenic action. The determination of the growth of the levator ani muscle in castrated rats was found to be a useful screening procedure provided certain precautions are taken[15]. The weight of other muscles has also been used as an indication of anabolic activity, whereas the growth of the seminal vesicle, the prostate and Cooper's glands of castrated rats depends on the androgenic action. In the experimental animal, therefore, it is possible to differentiate between the two actions of any given compound.

A number of substances were tested, and a few were found suitable for clinical trials. In the early fifties Methylandrostenediol was used rather extensively[16, 17, 18]. The separation of the anabolic from the androgenic action, however, was not effective enough and also it was too expensive.

Nilevar*

17 - Ethyl - 19 Nortestosterone is an anabolizer which has been used widely during the past four years, and a number of clinical balance studies have been published. The literature dealing with this compound is voluminous, and a few points appear to be controversial at the present time.

* For simplicity, the trade names will be used in the remainder of the discussion.

A nitrogen sparing effect has been demonstrated by all investigators. In addition the excretion of Calcium in urine and in faeces is diminished. The retention of Phosphorus is proportional to the N and Ca retention. The retention of water, sodium, potassium and chlorides may relatively exceed the N retention and is not accompanied by additional weight gain. The positive fluid and electrolyte balance may be accounted for on the basis of increased perspiration[19]. The first investigators used a rather high dosage—50 mg. daily—and demonstrated some androgenic properties. A five year old boy who was treated over a six months' period for fragilitas ossium showed visible growth of pubic hair and penile enlargement[20]. The same authors found that the calcium retention continued for the six months' period, whereas the effect on Nitrogen retention appeared to decrease during the prolonged administration.

In a carefully conducted study it was determined that the increase in body weight is accompanied by a small increase in total body water and insignificant changes in extracellular water[21]. It was concluded that the weight gain is due to an increase in lean body mass and not due to water retention. In the same paper the authors suggest a previously underweight patient will maintain the weight after cessation of therapy, whereas a well nourished individual will lose the additional weight again. Our own experience would appear to indicate that this is not generally true and that some even grossly underweight patients will start to lose weight again as soon as therapy is discontinued.

The surgeons were interested in these substances from the beginning. Peden et al[22] reported their findings on 15 patients undergoing abdominal hysterectomy who were kept on a fixed caloric intake. The negative Nitrogen balance which commonly occurs during the postoperative phase was converted into a positive one by the administration of the drug, and the maximum effect was obtained by giving it both before and after operation. A considerable retention of sodium was also found in these patients. In the discussion of the paper Dr. W. E. Abbott[23] made three important points:

1. The sodium retention can be minimized by reducing the intake to 40-70 mEq daily.
2. The nitrogen balance is positive provided enough supporting calories are given in the diet.
3. It is easier to prevent nitrogen losses in females than in males.

In children Nilevar was found to enhance the growth rate[24]. However, since during long-term therapy there may be some androgenic effects (see above), its use is rarely justified.

Underweight persons gain weight if they take Nilevar on a long-term basis, as shown by Watson et al[25]. In order to exclude a possible psychogenic effect a control group received placebos and there was no weight gain in the latter group. This report suggests that the drug increases the body mass

either by stimulating some metabolic processes or by improving the appetite. The exact mechanism so far has not been determined. The dosage used was 25 or 50 mg. daily. The weight gain achieved was similar, but there were more side effects with the higher dosage.

Nilevar promotes fracture healing as shown in experiments with animals by the increased uptake of radioactive sulphur at the fracture site[26, 27]. It was postulated that it stimulates the synthesis of mucopolysaccharides in the new formed collagen tissue.

A beneficial effect of Nilevar on wound-healing has been reported by MacKenzie[28, 29].

An increased excretion of creatine was found by several authors and Dowben[30] concluded that this may reflect an increased creatine synthesis.

The side effects of Nilevar are rather important and need to be discussed in detail. The presence of an androgenic effect has already been mentioned. The androgenicity was especially noticeable in prepuberta! and hypogonadal males[31]. Following a rather high dosage for three weeks, five infertile men became impotent and the sperm count in the seminal fluid decreased[32]. Prolonged administration (8-10 weeks) in normal male volunteers (inmates of a penitentiary) was found to lead to azoospermia and complete loss of libido. After cessation of therapy the sperm count returned to normal or more and the libido also returned[33]. The urinary gonadotrophins are depressed[32, 33]. In the female the treatment may be accompanied by menstrual irregularities[31], and transient acne[32]. Menopausal symptoms are not affected[32]. In rats the estrous cycle can be inhibited[34, 35].

The effect of Nilevar on the adrenal cortex has been studied by several authors. The excretion of 17-ketosteroids is decreased[33, 36] and a suppression of adreno-cortical secretion was thought to occur[37]. However, Carter et al[38] showed that the adrenal response to ACTH is not altered by the administration of Nilevar and they concluded that there is no alteration in the adrenocortical function as measured by the 17-hydroxy-corticosteroid levels in the plasma.

Foss[39] states without elaborating the point further that testosterone as well as the less virilizing hormones should not be given to men with carcinoma of the prostate. On the other hand, Brendler and Winkler[36] found no effect of Nilevar on the course of prostatic carcinoma in five patients.

An important complication is the occurrence of jaundice following the administration of Nilevar[40]. Kory[41] found an abnormal Bromsulphalein retention in 74% of his patients. Other liver function tests were normal, and there was no clinical evidence of liver dysfunction. He postulated that Nilevar may inhibit specifically the transfer of BSP from the blood into the bile and that the liver dysfunction is mild and reversible. Schaffner et al[42] found histological evidence of cholestasis. A similar pattern has been observed following the administration of methyl testosterone[40, 43].

Other effects of Nilevar have been noted, but are not yet fully investigated. In chronically underweight individuals the blood cholesterol, phospholipids and lipoproteins were found to be decreased by Meade et al[44]. An attempt was made to diminish the urea production in patients with acute renal failure, by suppressing the protein catabolism with Nilevar[45]. The calculated mean daily urea production dropped in the cases of renal failure due to obstetrical causes, but was not significantly altered in others. Based on the assumption that Nilevar may strengthen the levator ani muscle in humans Nilevar was given to incontinent mental patients[46], but without lasting results.

Other papers dealing with purely physiological observations without practical application are not included in this review.

New Compounds

Recently two new substances have been developed, which are supposed to have a better separation of androgenic and anabolic action, Nandrolone phenpropionate (Durabolin^{R}, Organon) and Methandrostenalone (Dianabol^{R}, Ciba). The anabolic effect is said to be achieved with a smaller dosage than with Nilevar (Durabolin 25-50 mg. I.M. per week, Dianabol 5 mg. b.i.d. orally) without virilizing effects. Both of these drugs are not yet as well investigated as Nilevar.

Desaulles et al[47] published a pharmacological study employing the same experimental methods as outlined above and compared Dianabol with Testosterone and Nilevar. Dianabol provided a better separation of the anabolic effect from the androgenic effect, and changes in the estrous cycle in rats appeared only after a much higher dosage. In order to suppress the pituitary gonadotrophins again a much higher dosage was necessary. Whereas testosterone produced a greater weight gain in young male rats than in young female rats and whereas Nilevar was more effective in females, (see Abbott[23]) there was no sex difference following the administration of Dianabol. Part of the anabolic action seems to depend on the presence of a functioning pituitary gland. Dianabol is absorbed well from the gastrointestinal tract and can be given orally. Dianabol reduces the deposition of lipids in the arteries and liver of rabbits. In patients suffering from mammary carcinoma higher than the recommended dosage did not produce androgenic effects[48].

Based on scanty reports in the literature[49, 50, 51] Durabolin appears to have similar actions. Pharmacological observations on rats were reported by Overbeek and deVisser[52].

Durabolin also resembles Dianabol in its effect on the lipid deposition in the arterial wall. In cockerels the serum cholesterol level was found to be lower and there was a lower incidence of lesions in the coronary arteries[53].

C. Clinical Application

In practice the anabolic agents are used in conditions associated with excess protein breakdown or lack of sufficient protein retention. Such conditions are associated with a variety of disease and are not infrequently observed in geriatric patients. In extreme cases one speaks of cachexia. Lesser degrees occur more often and are termed asthenia, chronic debility states, undernutrition, underweight, etc. The restoration of physical health is an important part of rehabilitation and anabolic compounds are valuable adjuncts to the treatment. They are not more than adjuncts, however, and replace by no means the other therapeutic measures which are discussed in the other papers. Undernourished patients do not feel well. Because they do not feel well they do not eat enough—sometimes a vicious circle is set up which is difficult to overcome. Carefully prepared food served in an appetizing way in pleasant surroundings help many patients overcome their aversion to eating, and some do benefit from the addition of an anabolizer. It should be pointed out again that an anabolizer is valueless in the absence of an adequate food intake. After a while many patients regain normal eating habits and then do not need the drugs any more. Whether the anabolizer acts directly or indirectly on the patient's appetite is of no significance in this connection.

Bedridden, undernourished elderly patients often are suffering from bedsores. In the healing of an ulcer the "processes by which a cell takes from the blood the substance required for repair and growth" surely play an important part, and in practice the presence of bedsores is one of the most frequent indications for the administration of an anabolizer.

In the presence of an adequate dietary intake the addition of other supplements such as vitamin preparations is rarely justified. There is no harm in allowing the patient to have an occasional bottle of beer or a drink of his favorite whisky. Many of these undernourished elderly patients show evidence also of a hypochromic anemia, probably on a nutritional basis. The anemia is not influenced by the anabolizers, in fact in some patients we observed a drop of the hemoglobin concentration and of the hematocrit during the therapy. This may be a dilutional effect associated with an increase in circulating blood volume.

It may be added here that in the re-establishment of good eating habits the regulation of the bowels also is important. At the present time we use mainly Dulcolax[54, 55] for the treatment of chronic constipation.

Iatrogenic catabolism, a side effect often produced by prolonged administration of cortisone and similar drugs may in part be corrected by the simultaneous administration of an anabolizer, apparently without jeopardizing the clinical effect of cortisone[56].

The use in mammary carcinoma has been mentioned. When bone metastases are present the decreased calcium excretion may be desirable. The effect on bone pain possibly can be explained on this basis. Improvement of osteoporosis has been reported[49], but our own results in two cases of senile osteoporosis have not been striking.

More controversial is the administration of an anabolizer pre-and post-operatively. As already mentioned by Albright, the short catabolic phase following an operation or trauma may not be deleterious to the patient, on the contrary, it may set the stage for following normal convalescence. Through general catabolism the products necessary for repair at the site of injury may be made available. The value of anabolic substances during or before this phase is therefore questionable. In the majority of patients it is unnecessary. If for one reason or another the convalescence is prolonged, e.g. after severe burns, and protein breakdown takes place over long periods, the situation is different and anabolizers certainly are of value in these more chronic cases.

Other possible indications, such as uremia, metabolic bone disease, myopathies, retarded growth are beyond the scope of this paper. At the Municipal Hospital, Dianabol is under investigation. Thirteen patients have taken the drug regularly for a sufficient length of time. Seven patients showed a satisfactory response, five did not gain weight and in one the result cannot be evaluated because tube feedings were started at the same time. The Bromsulphalein excretion was determined in six patients after they had taken the drug regularly for three weeks. In three the test was negative and in three more than 5% retention was found. Some of the patients who did not respond were given 10 mg. b.i.d. or t.i.d., but again no effect was noted. One patient with chronic rheumatoid arthritis who did not respond to Dianabol, also failed to benefit from Nilevar. Clinical virilizing effects were absent in all patients.

The patients who responded favorably to the drug showed the usual gain in weight, improved healing tendency of bedsores, improved appetite and general well-being. Since this is not a scientifically conducted study not many conclusions can be drawn. The desired anabolic effect was present in half the patients. Bansi[57] reported similar results but a higher proportion of successful cases.

The alleged effect of Durabolin on calcium excretion has not been confirmed in a study on respirator patients. Preliminary results indicate that the calcium excretion remained unchanged during the administration of Durabolin[58].

Summary

The development and the present ideas regarding the use of anabolic substances have been discussed. Chronologically the first compound to be used as an anabolizer was testosterone. The application is limited because of the androgenic effect. Later

derivatives were found which have a similar anabolic and far less androgenic action. The main indication for clinical application are chronic debility, underweight and lack of appetite. Since some of these compounds have important side effects and others are not too well investigated yet, they are to be used with caution. Anabolizers are potent drugs and should only be used in the presence of a definite indication.

References

1. Taber, C. W.: Cyclopedic Medical Dictionary, 8th Ed. F. A. Davis Co., Philadelphia, 1958.
2. Kochakion, C. D. and Murlin, J. R.: The effect of male hormone on the protein and energy metabolism of castrated dogs. J. Nutrition. 10: 437, 1935.
3. Masson, P.: Deux cancers Leydigiens de l'homme. Rev. Canad. Biol. 2: 168, 1943.
4. Albright, Fuiler: Cushing's Syndrome. The Harvey Lectures. 1942-43, p. 123-186.
5. Caraballeira, A., Elrick, H., Mackenzie, K. R. & Browne, J. S. L.: Effects of single intravenous injections of pituitary growth hormone to normal adult men. Proc. Soc. Exp. Biol. Med. 81: 15, 1952.
6. Prudden, J. F., Young, M. K., Stirman, J. A.: Studies on Growth Hormone I. The effects of growth hormone and insulin upon the synthesis of protein in perfused livers. J. Lab. Clin. Med. 47: 270, 1956.
7. Prudden, J. P., Pearson, E. and Soroff, H. S.: Studies on growth hormone II. The effect on nitrogen metabolism of severely burned patients. S.G.O. 102: 695, 1956.
8. Pearson, E., Soroff, H. S. and Prudden, J. F.: Studies on growth hormone. V. Effect on the mineral and nitrogen balances of burned patients. Am. J. Med. Sci., 239: 17, 1960.
9. Gurup, F. E., Schwartz, M. S., and Prudden, J. F.: Studies on Growth Hormone. VI. Dependence of anabolism on the level of intake. Am. J. Med. Sci. 239: 27, 1960.
10. Kenyon, A. T., Sandiford, I., Bryan, A. H., Knowlton, K. and Koch, F. C.: The effect of testosterone propionate on nitrogen, electrolyte water and energy metabolism in eunuchoidism. Endocrinol. 23: 135, 1938.
11. Kenyon, A. T., Knowlton, K., Sandiford, I., Koch, F. C. and Lotwin, G.: A comparative study of the metabolic effects of testosterone propionate in normal men and women and in eunuchoidism. Endocrinol. 26: 26, 1940.
12. Kenyon, A. T., Knowlton, K., Lotwin, G. and Sandiford, I.: Metabolic response of aged men to testosterone propionate. J. Clin. Endocrinol. 2: 690, 1942.
13. Webster, B. and Hoskins, W.: Influence of androgen therapy on growth rate of hypogonadal adolescent boys. Proc. Soc. Exp. Biol. Med. 45: 72, 1940.
14. Cooper, I. S., Rynearson, E. H. and MacCarty, C. S.: Testosterone propionate as a nitrogen-sparing agent after spinal cord injury. J.A.M.A. 145: 549, 1951.
15. Gordan, G. S.: Experimental basis for anabolic therapy. Arch. Int. Med. 100: 744, 1957.
16. Gordan, G. S., Eisenberg, E., Moon, H. D. and Sakamoto, W.: Methylandrostenediol: a protein-anabolic steroid with little androgen activity. J. Clin. Endocrinol. 11: 209, 1951.
17. Querido, A., Kasenaar, A., Schuurs, M. A. M. and Seldenrath, H. C.: A balance study of methylandrostenediol in a patient with hypogonadism. J. Clin. Endocrinol. Metab. 12: 1077, 1952.
18. Homburger, F., Kasdon, S. H. and Fishman, W. H.: Methylandrostenediol: a non-virilizing derivative of testosterone in metastatic cancer of the breast. Proc. Soc. Exp. Biol. Med. 74: 162, 1950.
19. McSwiney, R. R. and Prunty, F. T. G.: Metabolic effects of three testosterone derivatives including 17d-Ethyl-19-Nortestosterone J. Endocrinol. 16: 28, 1957.
20. Prunty, F. T. G., Brooks, R. V., Clayton, B. E. and McSwiney, R. R.: Some effects of 17d-Ethyl-19-Nortesterone in man. Proc. Roy. Soc. Med. 51: 557, 1958.
21. Woodford-Williams, E. and Webster, D.: An anabolic study with Norethandrolone in four elderly underweight males. B.M.J. II: 1447, 1958.
22. Peden, J. C., Maxwell, M. C. and Ohim, A.: Anabolic effect of a new synthetic steroid on nitrogen metabolism after operation. Arch. Surg. 75: 625, 1957.
23. Abbott, W. E.: Discussion of (22). Arch. Surg. 75: 629, 1957.
24. Bayer, L. M.: Growth observations during the administration of a new steroid (Nilevar). Stanford Med. Bull. 15: 306, 1957.
25. Watson, R. N., Bradley, M. H., Callahan, R., Peters, B. J. and Kory, R. C.: A six-month evaluation of an anabolic drug, Norethandrolone, in underweight persons. I. Weight gain. Am. J. Med. 26: 238, 1959.
26. Kowalewski, K.: Catabolic effect of dihydrotachysterol and anabolic action of 17-Ethyl-19-Nortestosterone on the uptake of radiosulphur in a healing fractured bone of the rat. A. Endocrinol. 28: 119, 1958.
27. Kowalewski, K.: Comparison of the effects of cortisone and certain anabolic-androgenic steroids on the uptake of radiosulphur in a healing fractured bone. Endocrinol. 62: 493, 1958.
28. Gouws, F., Silbermann, O. and MacKenzie, W. C.: The effect of 17-Etnyl-19-Nortestosterone (Nilevar) on healing of experimental wounds. Canad. J. Surg. 1: 362, 1958.
29. Pisesky, W., Williams, H. T. G. and MacKenzie, W. C.: The effect of Triethylene thiophosphoramide (Thiotepa) and 17 - Ethyl - 19 - Nortestosterone (Nilevar) on wound healing. Can. J. Surg. 2: 291, 1959.
30. Dowben, R. M.: Increased creatine and creatinine excretion after 17 - Ethyl - 19 - Nortestosterone. Proc. Soc. Exp. Biol. Med. 98: 644, 1958.
31. Epstein, J. A., Vosburgh, L., Reid, G. and Kuppermann, H. S.: Clinical studies on Norethandrolone: an anabolic progestational steroid. Clin. Res. 5: 16, 1957.
32. Leach, R. B., Paulsen, C. A., Lauman, J., Goldston, N. W. and Maddock, W. O.: Norethandrolone: gonadotrophin suppression without androgenic or estrogenic activity. Clin. Res. 6: 261, 1958.
33. Heller, C. G., Laidlaw, W. M., Harvey, H. T. and Nelson, W. O.: Effects of progestational compounds on the reproductive processes of the human male. Ann. N.Y. Acad. Sc. 71: 649, 1958.
34. Richter, R. H. H. Die Wirkung von 17-Aethyl-19-Nortestosteron auf den oestrischen Zyklus der Ratte. Schw. Med. Wschr. 88: 1261, 1958.
35. Richter, R.: The effect of 17-Ethyl-19-Nortestosterone on the estrous cycle. Experientia. 14: 219, 1958.
36. Brendler, H. and Winkler, B. S.: Effect of Norethandrolone on 17-Ketosteroid excretion in prostatic cancer patients. J. Clin. Endocrinol. Metab., 19: 183, 1959.
37. Brooks, R. V. and Prunty, F. I. G.: The suppression of andrenocortical secretion with 17-Ethyl-19-Nortestosterone. J. Endocrinol. 15: 385, 1957.
38. Carter, A. C., Weisenfeld, S. and Goldner, M. G.: Failure of 17-Ethyl-19-Nortestosterone to effect plasma 17-Hydroxycorticosteroids and ACTH responsiveness in man. Proc. Soc. Exp. Biol. Med. 98: 593, 1958.
39. Foss, G. L.: The use of protein anabolizers. Brit. J. Clin. Pract. 13: 541, 1959.
40. Dunning, M. F.: Jaundice associated with Norethandrolone (Nilevar) administration. J.A.M.A. 167: 1242, 1958.
41. Kory, R. C., Bradley, M. H., Watson, R. N., Callahan, R. and Peters, B. J.: A six-month evaluation of an anabolic drug. Norethandrolone in underweight persons. Am. J. Med. 26: 243, 1959.
42. Schaffner, F., Popper, H. and Chestow, E.: Cholestasis produced by the administration of Norethandrolone. Am. J. Med. 26: 249, 1959.
43. Heaney, R. P. and Whedon, G. D.: Impairment of hepatic bromsulphalein clearance by two 17-substituted testosterones. J. Lab. Clin. Med. 52: 169, 1958.
44. Meade, R. C., Owenby, J. and Kory, R. C.: The effect of long-term administration of an anabolic drug, Norethandrolone on the blood lipids of chronically underweight individuals. J. Lab. Clin. Med. 50: 932, 1957.
45. McCracken, B. H. and Parsons, F. M.: Use of Nilevar to suppress protein catabolism in acute renal failure. Lancet II: 885, 1958.
46. Vlavianos, G. and Fink, L.: The clinical effect of nor-Eth.-Androlone (Nilevar) on incontinent mental patients. Am. J. Psychiat. 115: 164, 1958.
47. Desaulies, P. A., Krahenbuhl, Ch., Schuler, W. and Bein, H. J.: Etude experimentale du Dianabol R. un nouvel anabolisant. Schw. Med. Wschr. 89: 1313, 1959.
48. Hammerstein, J. and Gansau, H.: Probleme der Hormontherapie des Mamma-Karzinoms. Berliner Med. 10: 103, 1959.
49. Banghart, H. E.: Some Observations on Osteoporosis. Bull. Acad. Med. N.J. 5: 70, 1959.
50. Payne, R. W.: Anabolism, Appetite and Ambition. Journ. Kentucky Acad. G.P. Oct. 1959.
51. Szujewski, H. A.: Nandrolone Phenproprionate. A new palliative agent in the management of mammary carcinoma. Am. Pract. Digest Treat. 10: 2157, 1959.
52. Overbeek, G. A. and deVisser, J.: A. Endocrinol. 24: 209, 1957.
53. Campbell, R. S. F., Lawrie, T. D. V., McLaurin, J. C. and Pirrie, R.: The effect of 19-Norandrostenalone on experimentally induced atheroma in cockerels. Circ. Res. 8: 78, 1960.
54. Zingg, W.: Experiments with a new Laxative. Man. Med. Rev. 38: 460, 1958.
55. Pincock, J. G.: The use of a rectal suppository of Bisacodyl (Dulcolax) in geriatric patients. Can. Med. Ass. J. 82: 268, 1960.
56. Jeanneret, T., Esselier, A. F. and Morandi, L.: Nebennierenrinde, Mineral und Wasserhaushalt; Grundlagen und therapeutische Konsequenzen unter Berucksichtigung des Knochensystems. Schw. Med. Wschr. 87: 846, 1957.
57. Bansi, H. W.: Neue Ziele der Hormon-Behandlung. Med Klinik. 54: 673, 1959.
58. Hildes, J. A.: Unpublished results.

Pressure Sores

D. C. Brereton, M.D.

Pressure sores, or to use the alternative terms, bed sores or decubitus ulcers, are an ever-present problem in the chronic disease hospital. The medical staff must be constantly alert to the ease with which pressure sores may develop and so apply preventive measures at all times. Since about six percent of admissions to the Municipal Hospitals have pressure sores, their treatment becomes one of major importance on our wards.

It is the purpose of this paper to outline the mechanism of production of pressure sores and to discuss the classification and treatment.

The common denominator in the causation of these sores is pressure, usually over bony prominences such as the greater trochanters, sacrum, heels, etc. However, there are other factors which influence the development of pressure sores, such as the state of nutrition, chronicity of the illness, whether the nerve supply to the part is interrupted or intact, cleanliness of the skin and how often the patient can turn or be turned so as to relieve pressure areas. Pressure produces ischemia, and if unrelieved, necrosis.

It can be seen that many of these conditions are present in a chronic disease hospital. It is not surprising, in the chronically ill, bedridden patient who is either unable or unwilling to turn to other positions, who frequently soils himself with excreta, whose skin is altered from nervous disorder or senile change (or both) that ulcers may develop unless great care is taken to carry out the necessary prophylactic measures.

Groth[5] who produced experimental pressure sores in rabbits and studied the tissue pathology of the developing ulcers, drew several interesting conclusions. He pointed out that if pressure was applied at the skin surface over a bony prominence, the pressure on the skin was spread over a large surface, but the pressure over the bony prominence would be concentrated over a small area. Thus, there is a conical shaped pressure distribution and in this way it is possible to have a mild skin redness or necrosis, but deep to this there is increased necrosis of subcutaneous tissue, fat and muscle and hence the production of "the deep ulcer."

He also found that generalized sepsis could lead to infection at the site of pressure sores with abscess formation and spread of infection.

Different stages in the development of pressure sores are recognized. The following are those outlined by Campbell[1].

1. Redness of the skin over a pressure area.
2. Redness, swelling and induration with occasional blistering and desquamation of the epidermis.
3. Necrosis of skin with exposure of fat.
4. Skin and fat necrosis extending to muscle.
5. Skin, fat and muscle necrosis.
6. Bone involvement in the form of peri-ostitis, osteitis or osteomyelitis.
7. Osteomyelitis, septic arthritis, septicemia and possible death.

The usual locations of pressure sores in our hospitals are trochanteric and sacral, since most are chronic bedridden patients, many of whom have had cerebrovascular accidents, have deteriorated mentally and are incontinent. Some of the paraplegic patients also have pressure sores in the same situations. Some advanced multiple sclerosis cases have pressure sores on the heels, feet or ankles, depending where pressure is brought to bear by their contractures and aggravated by spasms. Pressure sores on the ears are not an altogether uncommon site, but usually respond well to treatment.

Regarding treatment, there are general measures which apply whether we are treating an existing pressure sore or whether we are trying to prevent one from developing.

1. Bring the patient to optimal nutritional state by high protein, high caloric, high vitamin diet. It is certainly not always an easy task to feed some of these chronic illness patients. Many are not hungry or interested in eating, many will not co-operate, and some have difficulty in swallowing—such as those who have had cerebrovascular accidents. Tube feeding, then, frequently becomes necessary. Nitrogen-retaining medications, such as testosterone propionate and nilevar, have been advocated. We have not been sure that such measures were of much benefit.

2. Anemia, if present, should be corrected by transfusions if necessary. Infections, also, must be dealt with in the appropriate manner.

General skin care is essential for the prevention as well as the treatment of pressure sores. The patient must be turned frequently in bed if unable to turn voluntarily. The judicious use of padding and rubber rings should be used in order to reduce pressure over vulnerable bony prominences. Gardner[2, 3] makes use of an alternating air pressure pad or mattress which reduces the amount of nursing time taken in turning patients. An ingenious German apparatus has been described by Neil[4] whereby the patient's pelvis is suspended by means of wires through the anterior superior iliac spines, thus taking pressure off sacral or pelvic sores.

Strict cleanliness of the skin is essential in order to prevent or heal pressure sores. The routine use of mild stimulating skin rubs, such as alcohol, has for years been recognized as a worthwhile procedure. Edema from congestive heart failure, hypoproteinemia or nephrosis, should be relieved if possible. Friction from sheets can be reduced by the use of talcum powder and protective clothing Creasing of sheets should be avoided.

For the treatment of the pressure sores of stages one and two, the application of the above principles of skin care is usually sufficient. If actual ulceration is present important measures include surgical removal of necrotic tissue and cleansing of the ulcer with various dressings, such as varidase (enzymatic debridement) and antibiotic ointments and gelfoam. Eusol dressings have been used over long periods of time in some chronic ulcers and seem to be satisfactory. Dressings are changed daily and more frequently than once daily if they become soiled.

Various surgical procedures for pressure sores have been widely practiced since World War II. It is generally thought that split-thickness grafting is not as satisfactory as closure with full-thickness flaps. Campbell[1] states that the object of surgery is to produce a well padded covering over the site of the sore and that this is best obtained by large flaps of skin and subcutaneous tissue. It has been found with split-thickness grafting that the grafts become adherent to the underlying bony prominence, with only a thin layer of intervening fibrous tissue, and that these ulcerated from unavoidable trauma to the denervated skin.

Since closure of these ulcers with a full-thickness flap, especially if the ulcer is large, produces some degree of shock, debilitated patients should not be subjected to this type of major surgical procedure. The healing is also delayed in the chronic, debilitated aged patient so that surgery should be pretty well limited to the younger age group. The majority of the patients in the Municipal Hospitals are not suitable candidates for surgical correction of their pressure sores.

References

1. Campbell, R. M.: Surg. Clin. of N.A. 39: 509, 1959.
2. Gardner: J.A.M.A. 138: 583, 1948.
3. Gardner: J.A.M.A. 154: 534, 1954.
4. Neil, J. F.: Apparatus for treating bed sores. Brit. Med. J. 2: 390, 1945.
5. Groth, K. E.: Clinical observations and experimental studies of Pathogenesis of Decubitus Ulcers. Acta Chirug. Scand. 87: 207, 1942.
6. Conn, H. P.: Current Therapy, 1956.
7. Crace, E. J. and Beakes, C. H. C.: The operative treatment of Decubitus Ulcer. New Eng. J. Med. 237: 141, 1947.
8. Stieglitz, E. J.: Geriatric Medicine. Lippincott Co., 1954.

♦ ♦ ♦

The Folly of the Foley

J. A. MacDonell, M.D.

Introduction

During infancy and early childhood, and in the "second" childhood, incontinence is an expected accompaniment of the age. However, where on the one hand an optimistic attitude is maintained and understanding with regular training are provided, on the other, unfortunately, the prevalent attitude is rather one of pessimism and of disinterested revulsion with the provision of somewhat punitive control measures.

Urinary incontinence constitutes a considerable, but certainly not insurmountable, problem in the care of the aged. However, when long-term indwelling catheters are used for the "control" of such incontinence, the problem rather than being solved is augmented. A sequence of pathological changes is needlessly initiated; increased demands for extra nursing care begin, and hope of obtaining rehabilitation of the patient receives an unjustifiably serious and perhaps permanent set-back. Thus, it is the purpose here to examine in more detail the effects of indwelling catheters and the mechanisms of urinary incontinence (where structural urinary tract abnormalities are not the cause) and to suggest alternate measures of achieving bladder control.

Catheter Effects

Bacteriological

Urinary tract infection is a frequent consequence of even a single catheterization. After the insertion of a catheter a positive culture was obtained on previously sterile bladder urine (using needle aspiration for sampling) in one third of cases[1]. When the catheter is left in place for prolonged periods the results are even more striking. Bacilluria developed within four days in 95% of cases in one series despite "adequate" precautions and prophylactic antibiotics[2]. Studies of the sources and routes of infection have provided some interesting observations. The same strain of infecting organism was demonstrated in the urine of all patients with indwelling catheters on a single hospital ward. That strain was also grown on culture plates exposed to the air in the ward and from attendants' hands, from towels and from Winchesters and syringes used on the ward[3]. As the catheter lies in the urethra, a mucopurulent sheath of exudate is formed around it. This sheath provides an admirable medium in which bacteria may proliferate and migrate from urethral meatus to bladder[4]. In addition, motile bacteria can ascend a moving fluid column, their rate of ascent being inversely proportional to the rate of flow of the fluid[5]. Thus, bacteria may be transferred from collecting bottle to bladder via the lumen of the inlying catheter and applying the same principle may even traverse the ureters to reach the kidneys. Where indwelling catheters are used, only by the application of the most rigorous technique can the incidence of chronic urinary tract infections be reduced[6].

On tissues

Soon after the insertion of an indwelling catheter, the tissues of the urethra and bladder react to the presence of this foreign body. In the urethra the inflammatory response is tissue edema and exudate production, to which infection soon contributes its increment. Peri-urethral abscesses and fistulae occasionally result. Fibrosis almost inevitably ensues producing strictures or converting the urethra into a dilated, inelastic tube. This latter is an important consideration in the short female urethra for at this stage, leakage of urine around the catheter results —the very incontinence which it was expected to correct is perpetuated by the device.

In the bladder the reaction to the catheter is frequently an intense, often hemorrhagic cystitis, later to be augmented by infection. The bladder ultimately becomes fibrosed, contracted and indistensible, particularly when continuous drainage has been used. Pressure of the dome of the contracted bladder upon the catheter tip may result in the formation of a diverticulum and even in perforation and peritonitis[7]. The normal valve-like mechanism provided by the intravesical portion of the ureter is disrupted by fibrosis in the area.

In a series of 100 consecutive autopsies, there was a statistically significant increase in urinary tract inflammatory changes in general and of acute and chronic pyelonephritis in particular amongst those patients who had indwelling catheters as compared with those who did not[8]. This has been a commonly recorded observation[9, 10, 11].

On the patient

In the early stages after its insertion, tissue irritation produced by the indwelling catheter results in patient restlessness and complaints of pain. The patient is "difficult" and sometimes even pulls out the catheter. The management of this situation is usually to re-insert the offending instrument and to increase sedation. Thus, on the one hand a contribution to morbid processes in the urinary tract is made, while on the other cortical appreciation is dulled and the hope of sensible control of incontinence is delayed. In the later stages, after the catheter has long been present, when the bladder is well contracted and chronic inflammation well established, the effects upon the patient of a small bladder capacity and of chronic infection with frequent exacerbations scarcely need to be described.

On the Ward Staff

The expenditure of hours of their working time irrigating and changing catheters; cleaning the collecting bottles; nursing patients through acute exacerbations of urinary tract infection—these are the effects of caring for the chronic catheter patient on a Ward Staff.

Mechanisms in Incontinence

The "inability to prevent the discharge of urine" or the "discharge of urine. . . in the normal way at intervals but involuntarily"[13]—in the above definition of incontinence lies a clue to the understanding of the mechanisms involved.

Under normal circumstances, the relationship between pressure-volume, bladder filling effects and the neural connections of that viscus are considered in discussions of micturition. Thus, when volition and intra-vesical pressure make their demands, the co-ordinated action of the parasympathetic controlled detrusor and the voluntary reciprocal nervous co-operation between the external sphincter and the abdominal muscles, produce bladder emptying[14]. The voluntary component is impaired or deranged in the geriatric patient by cerebrovascular disease, by acute illness and by the use of sedatives. Good muscle tone is likewise a prerequisite of efficient function. However, recumbency in removing the stimulus of maintaining an erect posture, drains the muscles of their tone, substance and strength. In addition, bedfastness produces apathy, the antithesis of voluntary effort[15]. The reduction of bladder capacity by a feces-loaded rectum, or the hypertonic bladder, complicate control of micturition. Hence, in this type of patient, either a defect in cortical executive correlation of receptor information and effector activation or inefficiently functioning muscles or a combination of both these factors, is responsible for the majority of cases of incontinence.

Thus, in the elderly patient, an appreciation of the causal factors and mechanisms permits the management of urinary incontinence to be conducted on a logical and rational basis.

Management of Incontinence

A realization of the clinical circumstances with which incontinence in the elderly is frequently associated—a stroke (large or small), an acute illness, confusion, drug depressed cerebration — and the knowledge that socially acceptable automatic bladder emptying can confidently be expected as a minimum attainment is the first requisite in the management of incontinence.

The Ward Staff is taught to observe the intervals at which expulsion of urine occurs. It then becomes a simple matter to keep the patient dry by regularly taking him to the toilet just before that time. This technique of toilet training, common practise in children, is less time consuming, ultimately, than caring for a catheter patient. The added dividend of a more comfortable, more confident, healthier patient whose continence improves with the regularity of the regime, is worth the investment of time and care. Nocturnal wetting sometimes lingers for long periods after diurnal dryness has been achieved. However here again, the expedients are simple and effective—waking the patient once or twice a night for toilet, or the provision of a diaper or condom drain.

All forms of voluntary and assisted muscle activity must be encouraged and bedfastness avoided if the necessary background of healthy muscle tone

is to be provided. Attention to bowel habit is essential. Encouragement with each success and sympathetic understanding of the "accidents" provide the incentive of moral support in the patient's effort to regain social independence. The availability of occupational therapy, ward group activities, anything to arouse the patient's interest, will prevent the lethargy which leads to careless disinterest in personal habits. These attitudes and methods are explained to the patient himself and to those who share the home environment to which he will ultimately return.

Discussion

Incontinence is common in the elderly infirm. Reduced bladder capacity, depressed cortical control and recumbency are frequent contributory causes. Since the resultant effect is amenable to relatively simple re-training measures, it seems illogical to use indwelling catheters for the control of such incontinence. Indeed, except in rare circumstances, it would appear that indwelling catheters are contra-indicated because of the pathological changes they produce in the urinary tract. Surely it is folly to use a Foley for simple incontinence.

Summary

The effects of indwelling catheters and the mechanisms contributing to incontinence have been described. The necessity of maintaining an optimistic and encouraging attitude towards the incontinent patient, and of the provision for him of a sensible routine including regular toilet times, attention to bowel habits, and physical and mental activity have been suggested.

Bibliography

1. Guze, L. B. and Beason, P. B.: Observations on Safety of Bladder Catheterization. New England J. Med. 255: 474, 1956.
2. Kass, E. H.: Asymptomatic Infections of the Urinary Tract. Tr. A. Am. Physicians. 69: 56, 1956.
3. Dutton, A. A. C. and Ralston, M.: Urinary tract infection in a Male Urological Ward. Lancet 1: 115, 1957.
4. Kass, E. H. and Schneiderman, L. H.: Entry of Bacteria into the Urinary Tracts of Patients with Inlying Catheters. New Eng. J. Med. 256: 556, 1957.
5. Weyrawch, H. M. and Bassett, B. J.: Ascending Infection in an Artificial Urinary Tract. Stanford M. Bull., 9: 25, 1951.
6. Miller, A., et al: Catheter Drainage and Infection. Lancet 1: 310, 1960.
7. Wallace, A. C. (Pathologist, Winnipeg Municipal Hospitals): Personal communication.
8. MacDonell, J. A. and MacDonell, J. A. K.: "The Case Against the Catheter," Paper presented at a Clinical Luncheon, Deer Lodge Hospital, May 1959.
9. Beeson, P. B.: Editorial. Am. J. Med. 24: 1, 1958.
10. MacDonald, R. A., et al: Relation between Pyelonephritis and Bacterial Counts in the Urine. New Eng. J. Med. 257: 915, 1957.
11. Beeson, P. B.: Factors in the Pathogenesis of Pyelonephritis. Yale J. Biol. and Med. 28: 81, 1955.
12. Kass, E. H.: The Management of Infections of the Urinary Tract. Am. J. Med. 18: 764, 1955.
13. Stedman, T. L.: Practical Medical Dictionary. Williams and Wilkins.
14. Swartz, D.: Neurogenic Bladder. C.M.A.J. 54: 333, 1946.
15. Baker, C.: Confined to Bed. Guy's Hosp. Gaz. 67: 11, 1953.

♦ ♦ ♦

The Hopeless Case

"Hopeless" patients are those of any age group who are suffering from a disease or state which we, with our present knowledge, regard as incurable. At any moment with increasing knowledge, one or more will be struck off the list. In other words, the incurability or hopelessness is used by us as an expedient pending further research, and there is no reason therefore why we should not adopt a positive approach to those patients from the very beginning, and follow the excellent example of the Bishop of Durham (Harland, 1957) who said of the dying that they must never be deprived of their last joy on earth—which is hope.

Hope is a state of mind in which one expects confidently that what one desires will come about—it is confident anticipation. Until a certain stage is reached in the lives of these patients for whom we have so far failed to find a cure, they are confident of their return to a state of health comparable to that of the average individual they see around them. They are, in fact, hopeful. When they begin to die their desires change, so that they look forward with confidence to their dying and to this world's release from their adversities. They are still hopeful.

Ronald Gibson: Proceedings of the Royal Society of Medicine, February, 1958

Observations on a Diphtheria Outbreak in Winnipeg

A. Cameron Wallace, M.D., Ellen Taylor, M.D.
Leslie Lansdown, M.D.

During the eleven month period from May 1956 to March 1957, an outbreak of diphtheria occurred in the city of Winnipeg. When the number of cases occurring, 66, is compared with epidemics of the recent past, the outbreak seems trivial. For example, the city of Amsterdam in 1944 recorded nearly 8,000 cases[9], and in Halifax, N.S. in 1940-41, 649 cases occurred among the civilian population[11]. Nonetheless, the pattern of this outbreak followed in miniature that of larger epidemics, and might have been far more serious. It is worthy of recording as an example of what can occur in a Canadian city at the present time, and may serve as a warning and guide to other communities. In addition, the clinical and pathological findings in fatal cases are recorded, because a search of the literature has revealed remarkably few such descriptions in the English medical literature.

A) Epidemiological

One fairly severe case of diphtheria and one carrier were reported in Winnipeg late in May 1956, but no cases were reported in June or July. The eventual distribution of cases is shown in Table 1.

Table 1
Cases — Month of Onset

1956	May	1 case		
	August	6 cases		
	September	3 cases		
	October	14 cases	Carriers:	
	November	21 cases	Male	7
	December	4 cases	Female	13
1957	January	8 cases		
	February	3 cases		
	March	6 cases		
	Total	66 cases (31 male, 35 female)		

Sixty-six cases in all occurred over a period of nine months with the largest number in November, 1956. Thirty-one cases occurred in males, and 35 in females. The experience of previous epidemics has been that where children are involved, males and females are equally affected; where large numbers of adults fall victims, as in the Halifax epidemic, females are in the majority, probably because of closer contact with children[9]. The age distribution of the Winnipeg cases is given in Table 2 and is compared with that of a few previous major epidemics. It can be seen that young children were the group most involved in this epidemic. One factor in this may be the restricted size of the outbreak; in epidemics such as those in Halifax and Amsterdam, the initial infections involved mainly children, but a gradual shift to the older age groups occurred. Possibly this might have been found in Winnipeg, had the disease spread more widely.

Table 2
Age Incidence of Diphtheria in Various Epidemics

	0 - 4	5 - 9	10 - 14	15 and over
Winnipeg, 1956-57	30.3%	51.5%	13.6%	4.6%
Baltimore, 1944	25.5%	46.5%	15%	13%
Halifax, 1940-41	14%	20%	14%	52%
Tyneside, 1941-42	11%	64%		25%
Amsterdam, 1942-47	26%	24%	20%	30%

Characteristic of most previously reported epidemics was the localization of the cases within the city. The map in Figure 1 demonstrates the concentration of most of the cases into a small area in the centre of the city. This area includes the poorest housing and economic conditions in Winnipeg, especially that adjacent to the C.P.R. freight yards. Similar concentration of cases was stressed in the reports of the Baltimore epidemic of 1944[4], and also in Amsterdam[9], and in the Newcastle-Gateshead

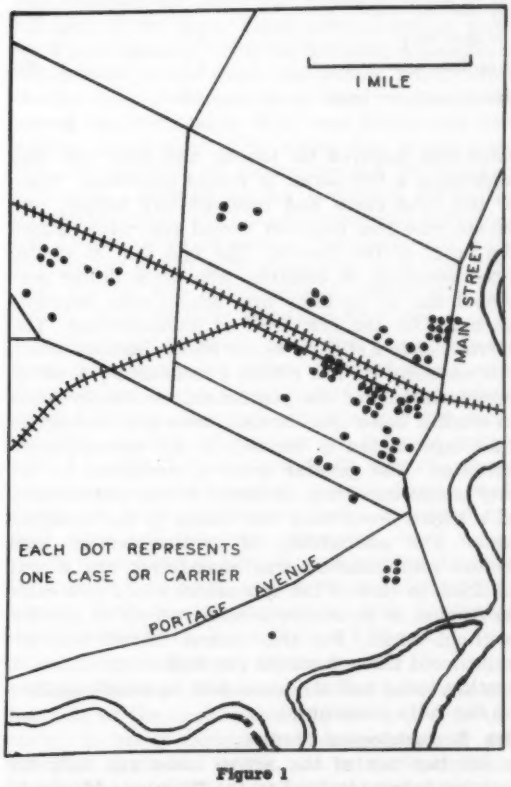


Figure 1

Map of central Winnipeg area showing location of cases and carriers. Note the concentration in the vicinity of C.P.R. freight yards, one of the poorest housing areas in Winnipeg.

From the Department of Pathology, Faculty of Medicine, University of Manitoba; Winnipeg Municipal Hospitals; and Provincial Laboratory, Province of Manitoba.

cases[5], where most of the outbreak was confined to crowded, poorly housed population. This concentration of the cases into a restricted zone is further emphasized by the incidence of multiple cases in one home, shown in Table 3. Of the 66 cases, 42 occurred along with one or more others in the same home, and 21 of the cases were centred in four homes. Carriers were found in the same areas, but only 20 carriers were identified.

Table 3
Incidence of Multiple Cases in One Home

Homes where:	Homes	Cases
One case occurred	24	24
Two cases occurred	10	20
Three cases occurred	1	3
Four cases occurred	2	8
Five cases occurred	1	5
Six cases occurred	1	6
	39	66

The immunization status of the cases studied is shown in Table 4. Three-quarters of the clinical

Table 4
Immunization Status

	Cases		Carriers	
No toxoid received	50	(75.8%)	12	(60%)
One dose only	4		....	
Two doses only	5	24.2%	1	
Three doses only	5		3	40%
Three doses + one booster	2		4	
	66		20	

cases had received no toxoid, and very few had undergone a full series of toxoid injections. None of the fatal cases had received any toxoid; one severe case had received toxoid one week before the onset of the disease. The fact that 35 of the cases occurred in children who were either preschool age or in their first school year accounts partially for the low level of immunization. The carriers gave a slightly better immunization record, but certainly do not reflect a satisfactory level of immunization for the population. Antibody levels in clinical cases and carriers were not studied, so that information is limited to the immunization histories. The general level of immunity in the city is also uncertain, although it was undoubtedly at a higher level than that found in the epidemic area. The advisability of routine Schick tests before immunization was considered, but it was decided, in view of the appearance of six new cases in August, to immunize as many people as possible without delay. For this reason, toxoid was administered throughout the city without prior Schick testing, using half the usual dose to avoid reactions in the fully immunized.

B) Bacteriological Studies.

All but one of the active cases and half the carriers were admitted to the Winnipeg Municipal Hospital. Bacteriological studies were made here, and duplicate material was sent to the Manitoba Provincial Laboratory. The main studies in both laboratories consisted of culture on Loeffler's medium with direct smear after approximately 24 hours, and further attempts to isolate the pathogen using tellurite plates. The Provincial Laboratory also received large numbers of swabs from contacts and from mass screening programs carried out in schools and other areas; in all about 1,500 specimens were examined. It soon became impossible to study all cases completely, particularly as regards virulence tests. All the virulence tests were done by the Provincial Laboratory, and guinea pigs were used exclusively. This test requires use of two guinea pigs for each case studied. The use of intradermal rabbit tests or in vitro tests might have made possible more extensive studies to verify routine bacteriological diagnosis.

In practice, where an isolated or initial case from an area was detected, virulence and biochemical tests were done on morphologically typical organisms. Where a definite contact for the case had been established, typical organisms on culture and smear were considered sufficient evidence for diagnosis. The diagnosis of a carrier was almost invariably verified by a virulence test, especially where a wage earner was involved.

All the organisms recovered in the epidemic showed starch fermentation and although some doubt remains as to the validity of this reaction for classifying diphtheria, it is felt that the entire epidemic was probably due to a gravis strain. No antigenic studies were done on the organisms; however, their value in classifying the organism is even more contentious than starch fermentation[10]. The morphologic observations on fatal cases were certainly consistent with gravis infections as described by others[7].

C) Clinical Observations

Of the 66 active cases, 31 were classified clinically as mild, 20 as moderate, and 15 as severe. All had been ill for at least two days before consulting a physician. Forty of the patients presented with the sole complaint of a sore throat, while headache accompanied this symptom in most of the other cases. Vomiting had occurred in four cases, and diarrhoea in one. One case had severe nosebleeds, and two suffered from difficulty in breathing on admission because of swelling of the throat.

Examination on admission revealed enlarged cervical nodes in 34 cases, and seven of the severe cases showed the classical "bull neck" of severe cervical edema. Twenty cases displayed some degree of nasal involvement, mainly a serous or serosanguinous discharge. Most cases had pharyngeal membrane, varying from a small streak to a heavy coating involving pharynx, tonsils and uvula. No typical laryngeal diphtheria as a distinct entity was admitted, although two fatal cases showed some laryngeal involvement at autopsy.

Treatment on admission consisted of diphtheria antitoxin, antibiotic therapy and such supportive

therapy as was indicated in individual cases. Carriers were given 10,000 units of antitoxin; active cases usually received 40,000 units. Serious cases were given 50,000 units; 5,000 to 20,000 of this was given intravenously depending on the age of the patient. Penicillin was used on the first cases treated, 500,000 units on admission and 1 cc. SR penicillin B.I.D. for four days. Later in the epidemic terramycin and chloromycetin were given to some of the patients. A tracheotomy was necessary with one patient because of edema of the throat.

Temperatures varied from 99° to 104° in the first 48 hours, and pulse was correspondingly elevated. Respiration rate was not elevated as a rule. In all but the severely ill patients, temperature and pulse fell within 48 hours of treatment and the membrane showed signs of clearing by the end of this period. The average hospital stay was 15 days, omitting in this average one patient who remained for 107 days. By reference to the records of previous outbreaks it can be shown that 15 days is a comparatively short average stay. The general condition of those patients leaving the hospital on discharge was satisfactory, except for two cases. These are discussed in detail elsewhere in this issue[6]. One of these was a three year old boy who was admitted with a severe pharyngeal diphtheria. Electrocardiographic abnormalities appeared and were still present on discharge eight weeks later. No evidence of heart failure was present. The other was a 13 year old girl who was gravely ill on admission with pharyngeal diphtheria. She developed palatal and anterior tibial paralysis, and her electrocardiogram showed marked abnormalities. Eight months later there was still an abnormal electrocardiogram, dyspnea on exertion and a persistent foot drop.

Four deaths occurred during the outbreak, all directly due to diphtheria. Table 5 shows the mortality of this epidemic in comparison with other

Table 5
Mortality from Diphtheria in Various Epidemics

	No. of Cases	No. of Deaths	Mortality
Winnipeg, 1956-57	66	4	6.1%
Baltimore, 1944 (4)	141	9	6.4%
Halifax, 1940-41 (11)	649	24	3.7%
Tyneside, 1941-42 (5)	264	9	3.8%
Amsterdam, 1944 (9)	7,932	182	2.3%

outbreaks. The small number of cases makes the comparison of limited significance. Nonetheless, the mortality (6.1%) is relatively high and emphasizes that diphtheria still is a serious disease. The Baltimore epidemic with a similar mortality was due to a minimus strain, while lower mortalities from a gravis strain were recorded in Halifax and at Newcastle and Gateshead. The Amsterdam figures shown represent only a small segment of a pandemic of diphtheria which raged for several years in Northwest Europe[1]. The average mortality in 1944 in Amsterdam was 2.3% but mortality from gravis strain was said to be 7.5%.

D) Description of Fatal Cases

The clinical and pathological features of the four fatal cases will be presented briefly. Three of these died and were autopsied at the Winnipeg Municipal Hospital. The fourth case is from the Children's Hospital, Winnipeg, and we are indebted to Dr. J. Hoogstraten for supplying us with the information and slides from this case.

Case 1

Clinical Summary

The first fatal case was that of L. S., a seven year old boy. He was admitted on September 20th, 1956, at 11 p.m., with a sore throat, difficulty in breathing and difficulty in swallowing. The boy was a recent immigrant from Germany, and had never received diphtheria toxoid. He had had a sore throat for five to seven days before admission, which had become gradually worse. He was not seen by a doctor until the day of admission, and by this time was seriously ill, with a foul coated pharynx and swelling of the neck. The family doctor suspected diphtheria and sent him to hospital.

On admission, temperature was 104.2°, pulse 107, respirations 24, B.P. 60/40. The child was dyspneic and cyanosed and there was severe swelling of the left side of the neck. Large cervical lymph nodes were palpable, mainly on the left side. A membrane was present on the uvula and tonsils, and C. diphtheriae was recovered from this, confirmed by guinea pig inoculation. WBC was 24,000 with 39% mature neutrophils, 17% stabs, 5% metamyelos, 34% lymphocytes, 5% monocytes and 2% degenerated cells. Because of his breathing difficulties oxygen was given and a tracheotomy was performed half an hour after admission. Antitoxin was given in a dose of 40,000 units along with penicillin. The next day he was still cyanosed, dehydrated and anuric; blood pressure remained very low.

In the next 24 hours concerted attempts were made to improve his severe state of shock with dextrose and saline, molar lactate, dextran, whole blood (100 cc.), Darrow's solution, and 150 mgm. cortisone. Serum sodium was 126 mEq./L., potassium 4.3 mEq./L. and chloride 95 mEq./L. Some improvement in his condition was noted and a small amount of urine was obtained; this contained red cells, casts and pus cells. However, chest x-ray, which was normal at first, showed mottled densities throughout both fields, and on the morning of September 22nd, he was unconscious with continuing shock and oliguria. He expired at 9.50 a.m.

Autopsy Summary

Gross Findings: There was marked edema of the left side of the neck, extending upwards into the scalp. A heavy necrotic membrane covered the pharynx, uvula and tonsils, and cylindrical masses of similar membranes were also present in the upper trachea. However, the airway below the tracheotomy was free from obstruction. The heart

was grossly normal. The lungs contained multiple zones of hemorrhagic consolidation, most extensive in the subpleural and dependent portions. The kidneys showed only swelling and congestion. The spleen was enlarged, and all the lymph nodes throughout the body were enlarged, soft and red. The remainder of the organs were essentially normal on gross examination.

Microscopic Findings: Sections of tonsils and pharynx showed extensive necrosis, edema and acute inflammation, all of which extended deeply into muscle (see Figure 2). Membranes covering

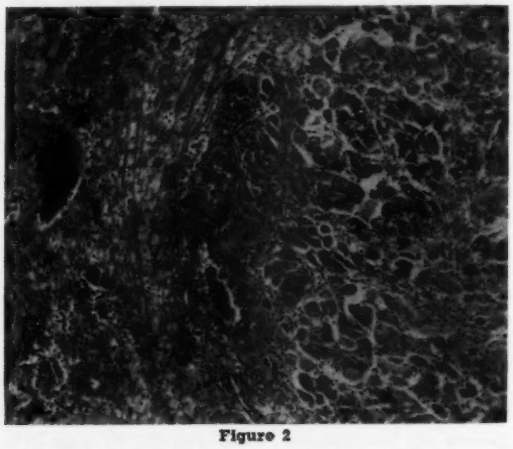


Figure 2

Section of pharynx from Case 1 at autopsy. Note necrosis of skeletal muscle, edema and fibrin deposition, heavy inflammatory exudate. Although the organism does not invade, inflammation extends deeply.

pharynx, tonsils, larynx and trachea contained organisms consistent with C. diphtheriae. Necrosis did not extend as deeply into laryngeal and tracheal walls. The lungs showed a severe acute bronchopneumonia accompanied by hemorrhage, congestion and edema. A mixed bacterial flora was present. Sections of heart revealed only a few small interstitial hemorrhages, not accompanied by necrosis or cellular infiltration. The kidneys showed typical lesions of ischemic tubular necrosis, consisting of severe diffuse bilateral destruction of tubular epithelium and accumulation of granular casts within the lumen. The adrenals showed minimal necrosis and small hemorrhages in the inner cortex. All of the lymphoid tissue throughout the body showed focal necrosis with a striking localization in the centre of the germinal follicles, replacing these areas with small masses of granular debris (Figure 3). This type of lesion was found in other autopsied cases in the present studies, and reference to older published pathological reports reveal that this type of lesion, although by no means specific for diphtheria, is quite characteristic[2, 3, 8]. Other organs showed no noteworthy microscopic lesion beyond cerebral edema of moderate degree.

Summary of Anatomical Findings

Diphtheria involving tonsils, pharynx, larynx and trachea.
Hemorrhagic bronchopneumonia.
Ischemic tubular necrosis of kidneys.
Extensive necrosis of lymphoid tissue.
Hemorrhages in myocardium and adrenals.
Edema of brain.

Case 2

Clinical Summary

This case bears marked resemblance to the first. C.S., a four year old girl, was admitted on January 10th, 1957, in an extremely serious condition. She had been ill with a sore throat for only a few days and was a neglected child who was seen only accidentally by a physician. As far as can be determined, she had never received any toxoid.

On admission her condition was described as very "toxic." There was a purulent nasal discharge, and marked edema of the right neck and throat. The tonsils were covered with a dirty grey exudate; cultures of this grew C. diphtheriae. Cervical lymph nodes were enlarged. The heart rate was rapid, and there was a loud to and fro murmur. The lung fields were clear. Temperature was 100° on admission; Hb. was 16.3 gm., WBC was 21,000 with 60% neutrophils and 23% lymphocytes. Urine contained pus, granular casts, and a few red cells and bacteria.

Over the next two days her condition failed to improve despite all therapy, which included diphtheria antitoxin, 40,000 units on admission, penicillin, and supportive therapy similar to that given to Case 1. Her mucous membranes were noted to be oozing blood, and purpura appeared on the skin. Temperature fell to 96° and she expired on the morning of January 13th.

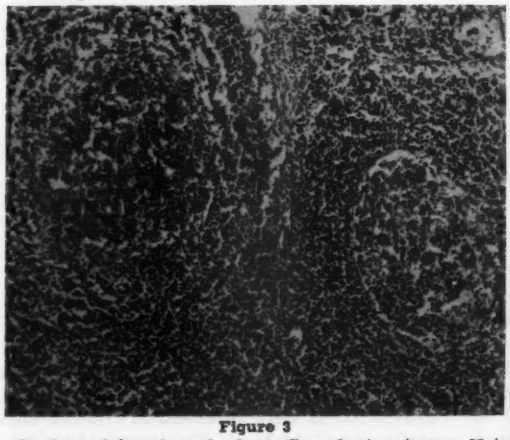


Figure 3

Section of lymph node from Case 1 at autopsy. Note necrosis of germinal centres of the follicles, leaving masses of deeply stained particulate material. This reaction was found in all four cases.

Autopsy Summary

There was severe swelling of the neck. The pharynx, tonsils and upper larynx were the seat

of a grossly apparent inflammatory process with necrosis, membrane formation and edema. The lower larynx and trachea were not involved, and no obstruction of the airway was apparent. Pulmonary edema and bronchopneumonia were present. The most striking gross finding otherwise, was the presence of severe hemorrhages involving all serosal and mucosal surfaces, the skin, lungs, adrenals and ovaries. The adrenal hemorrhages were minimal; the ovarian ones were very extensive.

Microscopically, in addition to local necrosis of a severe degree involving tonsils, pharynx and larynx, there was necrosis of the germinal centres of lymphoid tissue throughout the body, similar to that seen in the previous case. The myocardium contained small hemorrhages, and a few of these were associated with minimal focal necrosis, mainly subendocardial. These lesions were nowhere extensive. Sections of kidneys showed slight tubular necrosis.

Summary of Anatomical Findings

Diphtheria involving tonsils, pharynx and larynx.
Widespread hemorrhages involving serosal and mucosal surface, skin, lungs, ovaries and adrenals.
Pulmonary edema and bronchopneumonia.
Focal acute myocarditis, minimal.
Necrosis of germinal centres of lymphoid organs.
Tubular necrosis of kidneys.

Case 3

Clinical Summary

A. D., an eight year old girl was examined at the Out-patient Department of the Children's Hospital because of swelling of the neck and side of the face, accompanied by fever and vomiting. She had never received any toxoid. When first seen, a huge gray slough was found over the left tonsil and fauces, and bled when disturbed. Cultures, however, failed to reveal C. diphtheriae, and she was admitted with the diagnosis uncertain. She was not given antitoxin, but her temperature fell on penicillin and gantrasin, and her condition improved considerably in the next two days. On the morning of her third hospital day she quite suddenly became pale, cold and lethargic; apnea and coma ensued, and she died in a few hours.

Autopsy Summary

Some cervical swelling and lymph node enlargement was still present, but no definite membrane remained. The pharynx and tonsils were swollen and congested, but larynx and trachea were normal. The heart on gross examination was dilated and soft. The lungs showed only slight atelectasis.

Microscopically there was massive necrosis of tonsillar and pharyngeal mucosa, with extension of the process deep into underlying tissues. The spleen and lymphoid tissue throughout the body showed central follicular necrosis. Very small foci of myocardial necrosis with minimum inflammation were found, mainly beneath the endocardium. These were nowhere extensive. Small foci of hemorrhage and early necrosis were present in the left adrenal. There was no renal tubular necrosis.

Although C. diphtheriae was not isolated from this patient, the child was a known contact with two other proven cases, and the pathological picture was very similar to cases 1 and 2. In addition, Gram stains on a section of tonsil showed organisms suggestive of C. diphtheriae. For these reasons the case is considered to be one of diphtheria.

Summary of Anatomical Findings

Diphtheria involving tonsils, pharynx and upper larynx with necrosis involving underlying tissues
Necrosis of lymphoid follicles
Pulmonary atelectasis
Minimal acute myocarditis.

Case 4

Clinical Summary

V.N., an eight year old girl was admitted January 15th, 1957, seriously ill with a purulent nasal discharge and a soft, sloughing membrane on the tonsils. She had had a sore throat three weeks before admission, and there was a vague history of a rash one week later. She had never received any diphtheria toxoid. Her condition apparently became worse at home and she was admitted after her illness had been reported by a neighbor to the City Health Department.

On admission, in addition to the membrane and nasal discharge, she was noted to have enlarged, tender cervical nodes. Throat culture was positive for C. Diphtheriae. Lungs were clear on admission, but heart showed a gallop rhythm and was enlarged by x-ray. WBC was 13,000 with 62% neutrophils, 28% lymphocytes and 10% monocytes. Serum chlorides were 88 mEq./L., serum potassium was 5.35 mgm.%.

She was given 40,000 units of antitoxin and penicillin and some intravenous fluids were also given initially. Her condition remained poor and the child became increasingly listless. Vomiting and severe lower abdominal pain appeared two days after admission. Blood pressure fell to low levels and pulse became weak and irregular. She died two and one half days after admission.

Autopsy Summary

A very small amount of membrane remained in the nasopharynx, but oropharynx and larynx showed only congestion and C. diphtheriae was not isolated at post mortem. Bilateral pleural effusion and ascites were present, and the lungs showed atelectasis and congestion. All tissues were somewhat edematous. The heart was dilated but otherwise grossly not remarkable, and certainly not flabby or soft. The liver was markedly enlarged, weighing 800 gm.

Microscopically, the most striking feature was a severe diffuse myocarditis characterized by necrosis of variable duration, accompanied by heavy

infiltrate of lymphocytes, monocytes and neutrophils (Figure 4). In some areas these infiltrations were very heavy. The general picture was that of myocardial damage followed by inflammation and early repair, rather primarily an inflammatory process. The liver showed massive central congestion and necrosis, probably secondary to acute heart failure. Only slight inflammation was present in the nasopharynx. Lymphoid lesions as described for the other cases were present, but of lesser intensity. Lungs showed only slight congestion and atelectasis.

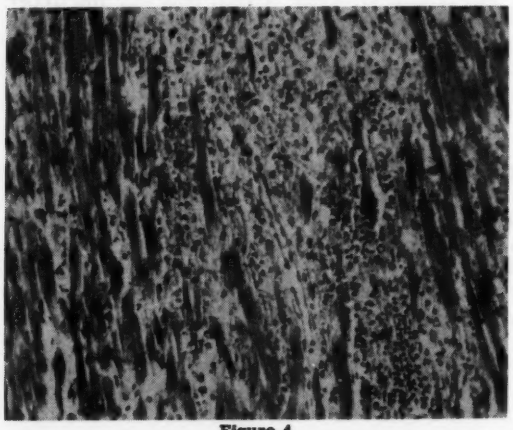


Figure 4

Section of myocardium from case 4. There is loss of myofibrils, giving the myocardium a moth-eaten appearance. A pleomorphic exudate is present, including lymphocytes, plasma cells, monocytes and neutrophils.

Summary of Anatomical Findings

Subsiding nasopharyngeal diphtheria.
Extensive diphtheritic myocarditis.
Acute hepatic congestion with massive necrosis.
Ascites, pleural effusion and edema of tissue.
Necrosis of lymphoid tissue.

Table 6
Summary of Autopsy Findings

	Case I Gravis	Case II Gravis	Case III ?	Case IV Gravis
Bacteriology				
Pharyngeal Involvement	++++	++++	++++	+
Laryngeal Involvement	+++	±	±	0
Myocarditis	±	±	±	++++
Lymphoid Necrosis	++++	+++	+++	++
Other	Hemorrhagic pneumonia; Renal tubular necrosis	Widespread hemorrhages; Renal tubular necrosis		Hepatic necrosis

Table 6 gives a recapitulation of the autopsy findings in the four fatal cases. It can be seen that three cases represented acute, fairly early infections with severe local lesions and death probably due to profound toxemia. Laryngeal involvement was marked only in the first case and did not lead to asphyxia. The fourth case represents a typical fatal myocarditis, appearing later in a severe case of diphtheria, causing death from heart failure at a time when the local lesions were on the wane. Lymphoid necrosis was a prominent feature in all but the fourth case, and probably represents a striking but non-specific result of severe toxemia.

Discussion

The epidemic we have described represents a limited but serious outbreak of diphtheria in a large community. It was apparently caused by a very virulent organism capable of producing a high mortality in the non-immunized. None of the fatal cases had received toxoid and none received antitoxin until the time of admission, when they were seriously ill. By this time the antitoxin administered probably had little effect.

There is little doubt that the epidemic could have spread much more widely had not a concerted effort been made to isolate carriers and cases, and to institute a city-wide immunization program. While no comparative figures are available, it seems likely that the overall level of immunity in the city was greatly raised by the immunization program over a period of a few months. It has been emphasized by epidemiologists that unless the proportion of immunized people in a general population is kept at a high level (about 75%), protection of a community against an epidemic cannot be assured[5]. Studies of previous epidemics have revealed that once a severe epidemic is established in non-immunized people, the disease may spread to involve the partially immunized, who, although they seldom suffer a serious attack, contribute to the problem of management and may spread the epidemic further[1, 5]. It should also be remembered that, in contrast to some other diseases, diphtheria immunity takes considerable time to develop after toxoid administration, and this makes the immediate control of an epidemic a more difficult problem. The task which must be achieved in every community is the maintenance of a high level of active immunization in a population to whom diphtheria is little more than a name and who are thus difficult to convince of the ever present threat of an epidemic.

Summary

An epidemic of diphtheria in the city of Winnipeg has been described. The main epidemiological, bacteriological, clinical and pathological features of the cases have been discussed, and compared with data from other epidemics. The outbreak had many of the typical features of previous epidemics, and the severity of the disease seems to have been equal to any reported in the recent past. The clinical and pathological features of four fatal cases are summarized.

Acknowledgment

We wish to thank Dr. Roper Cadham, Health Department, City of Winnipeg, for making available the epidemiological data.

References

1. Anderson, G. W.: Foreign and domestic trends in diphtheria. Am. J. Public Health, 37: 1-6, 1947.
2. Andrewes, F. W., Bulloch, W., Douglas, S. R., Dreyer, G., Gardner, A. D., Fildes, P., Ledingham, J. C. G., and Wolf, C. G. L.: Diphtheria. Med. Research Council London, H.M. Stationery Office, 1923.

3. Councilman, W. T., Mallory, F. B. and Pearce, R. M. A.: Study of the bacteriology and pathology of two hundred and twenty fatal cases of diphtheria. J. Boston Soc. M. Sc., 5: 137-319, 1900.
4. Eller, C. H and Frobisher, M.: An outbreak of diphtheria in Baltimore in 1944. Am. J. Hygiene, 42: 179-188, 1945.
5. Hartley, P., Tulloch, W. J., Anderson, M., Davidson, W. A., Grant, J., Jameson, W. M., Neubauer, C., Norton, R. and Robertson, G. H.: A study of diphtheria in two areas of Great Britain. Med. Research Council Special Report Series, No. 272, London, H.M. Stationery Office, 1950.
6. Israels, S. and Taylor, E.: Diphtheria myocarditis. Manitoba Med. Rev., Vol. 40: 363-369, 1960.
7. McLeod, J. W., Orr, J. W and Woodcock, H. E.: The morbid anatomy of Gravis, Intermedius and Mitis diphtheria. J. Path. and Bact., 48: 99-123, 1939.
8. Northrup, W. P.: Section on Diphtheria, Nothnagel's Practice Amer. Ed., Philadelphia, W. B. Saunders and Co., 1904.
9. Ruys, A. C. and Noordam, A. L.: The diphtheria epidemic in Amsterdam. Am. J. Public Health, 39: 185-194, 1949.
10. Wilson, G. S. and Miles, A. A.: Topley and Wilson's Principles of Bacteriology and Immunology. 4th ed., London, Arnold, 1955.
11. Wheeler, S. M. and Morton, A. R.: Epidemiological observations in the Halifax epidemic. Am. J. Public Health, 32: 947-956, 1942.

The Myocarditis of Diphtheria

Sydney Israels, M.D. and
E. Taylor, M.D.

This subject occupies fewer and fewer lines in each succeeding medical text. It is hardly believable that one could accumulate nine recent cases for recording in 1960, representing the dignosed cases of diphtheric myocarditis admitted to the Municipal Hospitals of Winnipeg from 1956 to 1959. During this same time, 86 patients with diphtheria were admitted to this institution. Details of an epidemic involved in this study are reported elsewhere in this issue.

Case Reports

1. A.H. Age 7½ years — male — admitted for a sore throat and swelling of the neck for a period of four days. Temperature 102° on admission, pulse 85, blood pressure 70/40. Culture of throat positive for virulent diphtheria.

Electrocardiographic findings showed depressed ST segments in lead 1, 2, V.F. and V. 4, 5, 6 and for biphasic T waves in leads 2, 3 and V.F.

Hemoglobin 16.3 grams on admission.

Treatment—penicillin and antitoxin.

Course—complaint of severe abdominal pain and collapse the day following admission. Blood pressure unobtainable. Child died.

Autopsy report—severe myocardial damage showing muscle necrosis and infiltration.

2. L.S. Age 7 years — male — sore throat and swollen neck one week, temperature 104.2°, tonsillar exudate.

No history of immunization.

Culture—virulent diphtheria.

Hemoglobin 14.4 grams. Serum NA 126 meq./L.

Chest X-ray — bronchopneumonia, normal heart size.

Treatment—antitoxin, penicillin, Solucortef.

Progress—severe abdominal pain, collapse.

Blood pressure 60/diastolic not obtainable. Anuria.

Autopsy — mild degree of myocardial infiltration and hemorrhage. Consistent with the diagnosis of diphtheritic myocarditis.

3. C.S. Age 4 years — female — sore throat and swelling of the neck two days. Tonsillar exudate noted.

Culture—diphtheria Vi positive.

Hemoglobin 16.3 grams.

Chest x-ray—negative.

E.K.G.—shows right bundle branch block, depressed ST segments in all leads and inverted T waves.

Blood pressure not obtainable.

Treatment—antitoxin, penicillin, cortisone.

Progress—collapse and death three days following admission.

Autopsy—myocardial necrosis and hemorrhage, and epicardial hemorrhage.

4. V.N. Age 5 years — female — sore throat 20 days. Tonsillar and vaginal exudate noted on admission. Pulse rate 140.

Culture positive for virulent diphtheria.

Hemoglobin 13.5 grams, serum NA 129 meq./L.

Chest X-ray—marked cardiac enlargement.

E.K.G.—low QRS voltage, depressed ST segments 2, 3 and V6.

Inverted T waves all leads.

Blood pressure not obtainable because the patient was in shock.

Treatment—antitoxin, penicillin, terramycin, cortisone.

Progress—severe abdominal pain, sudden collapse and death on third hospital day.

Autopsy — extensive myocardial necrosis, infiltration and hemorrhage.

5. D.D. Age 2 years—female—no immunization history, brought in because of fever and swollen neck. Examination showed tonsillar exudate.

Temperature 102.2°. Pulse rate 112.

Culture of throat positive for virulent diphtheria.

Hemoglobin 12.2 grams.

Chest X-ray—no heart enlargement.

E.K.G.—changes in the ST and T waves indicative of myocarditis, interventricular block, partial A.V. block.

Serum glutamic-oxaloacetic transaminase elevated 125 units.

Serum lactic dehydrogenase—elevated 570 units.

Treatment—penicillin, antitoxin, medrol.

Progress — child made gradual improvement and was discharged 41 days later. The transaminases and dehydrogenase had returned to normal but the E.K.G. showed abnormal T Waves.

Did not return for follow-up.

6. K.A. Age 3 years—female—admitted for sore throat. Examination on admission showed a tonsillar membrane.
Temperature 102°, pulse 90.
Throat culture virulent diphtheria.
Hemoglobin 14.0 grams.
Chest X-ray — normal heart size but evidence of bronchopneumonia. **E.K.G.** showed ST segment changes and T waves flattened in all limb leads and V.6.
Treatment—antitoxin, penicillin.
Progress — gradual improvement. Discharged 61 days later.
E.K.G. still abnormal.

7. L.A. Age 5 years—male—entered with complaint of sore throat for four days, and swelling of the neck. Examination showed a tonsillar exudate. Culture of throat grew virulent diphtheria organisms.
Heart rate 114.
Hemoglobin 12.6 grams.
Chest X-ray—normal. Serum NA 124 meq./L.
E.K.G. showed flattened T waves in all leads on the second day and S.T. segments were not remarkable but by the seventh day there was S.T. depression and T waves flattened in all leads.
Treatment—antitoxin and penicillin.
Progress — complaint of severe abdominal pain which gradually improved. She was discharged two months later and electrocardiogram was still abnormal. She did not return for a follow-up visit.

8. J.D. Age 30 years — male — sore throat three days, and large glands in the neck three days.
Physical examination showed a tonsillar exudate which grew virulent diphtheria organisms.
Blood pressure 128/80. Pulse 80.
Chest X-ray—normal.
Hemoglobin 18 grams.
E.K.G. showed ST and T wave changes in the limb leads and in V_2 and V_6 likely due to diphtheria.
Treatment—antitoxin, penicillin.
Progress—discharged in 14 days. Normal E.K.G.

9. J.M. Age 13 years — female — sore throat for three days and swelling of the neck for three days.
Examination showed membrane on both tonsils and culture of this grew virulent diphtheria organisms.
Pulse rate 100. Blood pressure 85/60.
Chest X-ray—normal.
Hemoglobin 14.4 grams.
E.K.G. showed low T waves in all leads. Considered to be abnormal and indicative of diphtheria myocarditis. It was abnormal by repetition as late as one month and six months thereafter.
Treatment—antitoxin and penicillin.
Progress — although the patient developed severe abdominal pain and vomiting, she gradually improved and was able to be discharged in one month still showing evidence of paralysis of her palate, which had cleared six months later when her electrocardiogram was still abnormal.

Discussion

None of the cases in this series had been immunized against diphtheria. All cases had positive throat cultures for virulent diphtheria organisms.

The finding of nine cases of myocarditis in 86 diphtheria admissions is close to the average of 10 to 25% usually found in diphtheria[1]. More cases likely existed, as all admissions did not have electrocardiograms or enzyme studies done.

The mortality of 45% closely approximates the usual mortality quoted[1]. This is stated to be 100% in children under two years of age, 50% in the two to ten year age group, and 25% in adults.

Severe abdominal pain was the most ominous symptom in the group studied. This is regarded by Friedberg[1] as a symptom of cardiac shock. The pain was so severe in some of the children as to simulate an acute abdominal catastrophe. Collapse accompanied this symptom in three of the patients who later died of their disease.

The electrocardiogram was abnormal in eight of the nine cases. The other child died before the test was done. ST and T wave changes were the most remarkable. Disturbances in conduction were also recorded in two instances. These changes could not be pursued in these children due to difficulty in getting follow-up records. However, in two, the electrocardiogram showed evidence of abnormality at least two months later and one six months later. Such damage may be long term as reported by Hoël and Sayers[2, 5] and Burkhardt[3]. The electrocardiogram was abnormal early in our cases, prior to the seventh day as usually stated[1].

Abnormal enzyme studies (transaminase and dehydrogenase) have been reported previously[4] and were present in one of our cases, returning to normal long in advance of the electrocardiogram.

Low levels of serum sodium were recorded in three of our severely ill patients. This likely is not specific for diphtheric myocarditis but is indicative of severe or prolonged disease.

The treatment of these cases seemed to make very little difference to the outcome.

Summary

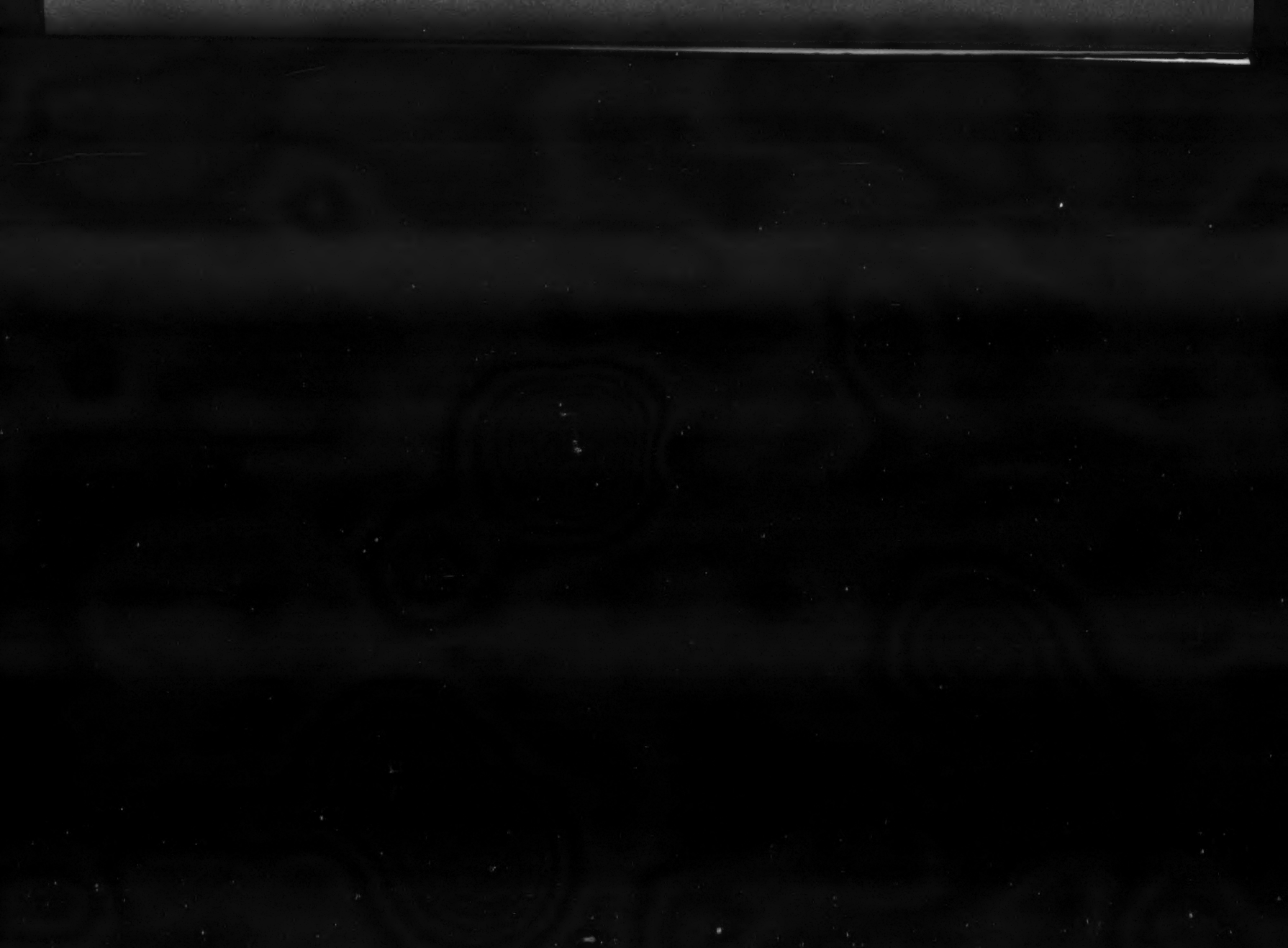
Nine cases of myocarditis of diphtheric origin are recorded.

Attention is called to the ominous symptom of severe abdominal pain preceding catastrophe. The cardiographic changes may persist for some time following the acute illness.

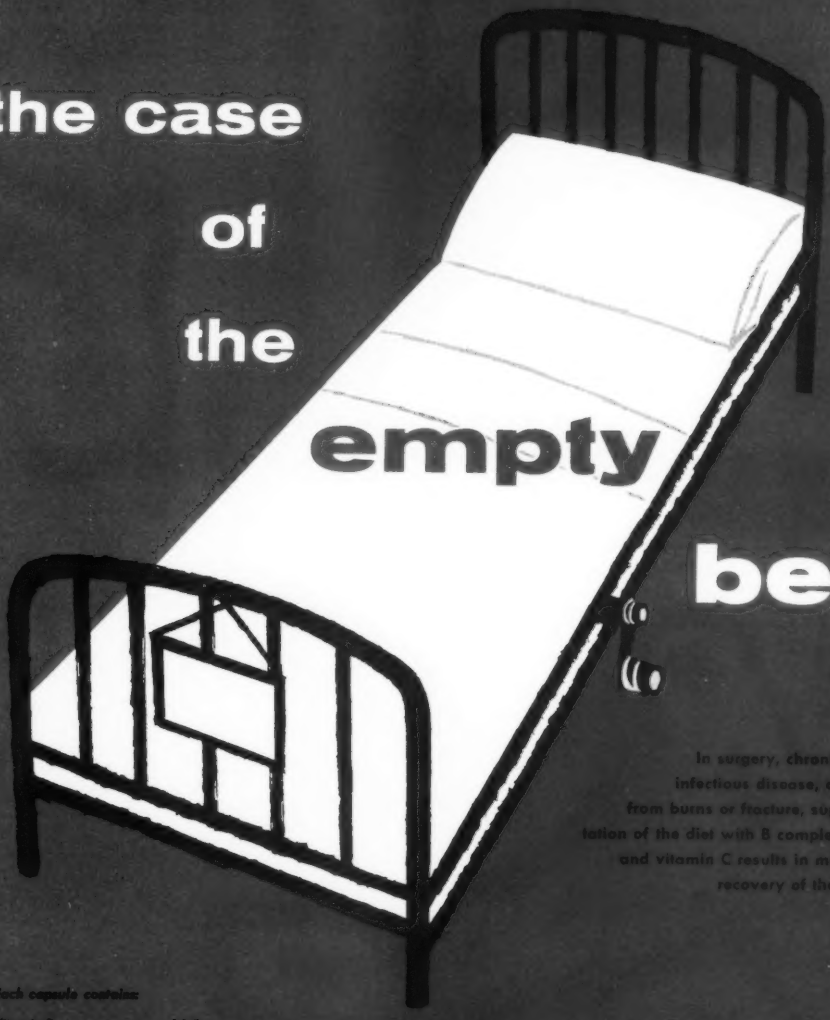
Finally, all the cases could likely have been prevented by proper immunization as none of the cases had received diphtheria toxin at any time.

Bibliography

1. Friedberg, A.: Diseases of the Heart. Saunders & Co., 2nd Ed., 1956.
2. Hoël, Jens: Acta Med. Scand. 237: 160, 1958.
3. Burkhardt, E. et al: Am. J. Med. Sc. 195: 301, 1938.
4. Chester, E.: Br. Heart Journal. 20: 24, 1958.
5. Sayers, E.: Annals Int. Med. 48: 147, 1958.



the case of the empty bed



In surgery, chronic illness, infectious disease, or trauma from burns or fracture, supplementation of the diet with B complex factors and vitamin C results in more rapid recovery of the patient.

Each capsule contains:

Vitamin B_{12}	10.0 mcgm.
Thiamine	25.0 mg.
Riboflavin	12.5 mg.
Niacinamide	50.0 mg.
Pyridoxine	1.0 mg.
Calcium d-Pantothenate	10.0 mg.
Vitamin C	250.0 mg.

One to three capsules daily.

"Beminal" with C *Fortis*

No. 817

Ayerst

Now available: **ELIXIR "BEMINAL" FORTIS No. 925**
nutritional tonic with taste appeal

Ayerst, McKenna & Harrison Limited—Montreal

B-6

Nutritional Macrocytic Megaloblastic Anemia

Leighton Young, M.D. and Sydney Israels, M.D.

A dietary deficiency of Vitamin B_{12} may produce hematological findings indistinguishable from those of classical pernicious anemia. One of the early descriptions of this deficiency was by Lucy Wills from India[1, 2]. Although most of the patients in her account were pregnant women, the tropical variety of nutritional macrocytic anemia, most frequently reported from India, Malaya, China and Macedonia, has also been recognized in non-pregnant women and in men[3]. In this particular group of cases it is appreciated that a simple Vitamin B_{12} deficiency may not be the sole cause, since many other factors are often also involved.

In temperate zones, macrocytic anemia resulting from dietary deficiency may also occur, but the condition appears to be less common than in tropical areas. R. A. Kern[4] in 1931, stated "in rare instances an Addisonian type of blood picture may result in a patient whose diet is deficient in the extrinsic factor." He supported this statement with clinical material. Since, many case reports have appeared[5, 6, 7, 8, 9], the diagnosis being based for the most part on a history of deficient diet, a classical blood picture and the therapeutic response to crude liver preparations. Most authors remarked on the deficiency of protein in their patients' diet. The one outstanding difference between these cases and the classical pernicious anemia is that most of the patients demonstrated free hydrochloric acid in their gastric juice. In those with achlorhydria at the time of diagnosis, the hydrochloric acid returned following treatment.

In a review of the literature only one case report of a nutritional macrocytic megaloblastic anemia, due to a dietary deficiency of Vitamin B_{12} but with histamine fast achlorhydria was found. The observations recorded below describe a second similar case of macrocytic anemia.

Case Report

This Manchester born spinster was admitted, at the age of 85, to the Winnipeg Municipal Hospitals on November 24, 1955 with the following history:

In 1925 she developed varicose veins. During 1940 recurring varicose ulcers made their appearance. In 1950 she was hospitalized for an unsuccessful skin grafting of chronic varicose ulcers. During this hospitalization she was given five blood transfusions for anemia.

On admission to the Winnipeg Municipal Hospitals examination revealed osteoarthritis of both knees with marked limitation of movement, extensive ulceration of the right lower leg with infection and congestive heart failure with a B.P. of 135/85. E.C.G. revealed an old infarction and chest x-ray, left ventricular hypertrophy. Hbg. was 14.8 gms%. From admission, she complained of nausea and vomiting and could be persuaded to eat little more than tea, toast and porridge. Radiological examination of her G.I. system revealed a large paraesophageal hiatus hernia producing pressure on the lower end of the esophagus which was dilated through the remainder of its length. She was digitalized, with improvement of her congestive heart failure. She received local therapy for her leg ulcers.

On August 9, 1956 the patient developed vomiting which persisted for 24 hours. She appeared pale and lapsed into semi-stupor. The following are results of pertinent investigation carried out at this time:

Blood: Hgb. 66.6 gms%; R.B.C. 1,660,000; Hematocrit 18.5; W.B.C. 1750; Platelets 98,000; Color Index 1.28, M.C.V. 104; M.C.H. 32.5; M.C.C. 17.

Peripheral Blood Smear: Marked anisocytosis and poikilocytosis. Macrocytes and moderate hyperchromia. Tendency to hypersegmentation of neutrophils.

Bone Marrow: Large numbers of cells suggestive of megaloblasts—typical of pernicious anemia.

Gastric Analysis: No free HC1 after histamine.

Stools: Negative for occult blood.

Schilling Test: Normal (10.6%).

The patient was provided with a high caloric intake via a feeding gastric tube. She was given 450 ccs. of packed red cells over a 36 hour period. Initial Vitamin B_{12} therapy consisted of 30 micrograms I.M. daily with a maximum reticulocyte response of 10.3% on the fourth day of treatment.

By the end of October 1956 her clinical condition had improved considerably. The following laboratory results were obtained:

Blood: Hbg. 12.0 gms%; R.B.C. 3,600,000; Color index 1.06.

Gastric Analysis: No free HC1 after histamine.

I.M Vitamin B_{12} was now given twice weekly. Her dietary habits were unchanged. Oral vitamin supplements which included B_{12} and folic acid were provided.

In October 1958 the Vitamin B_{12} was discontinued. By July 1959, the following laboratory results were obtained:

Blood: Hbg. 8.7 gms%.

Peripheral Blood Smear: Macrocytosis.

Bone Marrow: Normal.

Practical Plasma Proteins: Normal.

Serum Iron: 50 micrograms%.

B_{12} therapy was re-instituted and by Sept. 1959 the Hbg. had risen to 10.3 gms. Her Hbg. has remained at approximately that level to the present time

Leighton Young: Formerly Interne Winnipeg Municipal Hospitals. Present Address, Department of Pharmacology and Therapeutics, University of Manitoba.
Sydney Israels: Staff Physician, Winnipeg Municipal Hospitals.

despite the administration of both iron and Vitamin B_{12}. During this period, however, there has been chronic infection of her ulcerated leg and she has experienced two pneumonic episodes and recurring cystitis.

Comment

Castle has divided the nutritional anemias into two distinct types — direct or primary and conditioned or secondary. With the former, an anemia develops because of a dietary deficiency, while with the latter, the organism is unable to utilize a component of a balanced diet. Most "primary" deficiency anemias are due to an iron deficiency and are hypochromic microcytic in character. On the other hand, most secondary deficiency anemias are macrocytic in character and the most familiar of these is Addisonian pernicious anemia. The term "pernicious anemia" should be reserved for those cases due to a specific lack of Castle's intrinsic factor and should not be applied to the nutritional macrocytic anemias, as is sometimes done[8, 11].

It is now well known that Vitamin B_{12} deficiency in man gives rise to certain manifestations described in this case report. There are probably a number of mechanisms by which this deficiency may occur. These include[12]:

1. Deficiency of Castle's gastric intrinsic factor.
2. Bacterial or parasitic interference with normal absorption of Vitamin B_{12}.
3. Defective absorptive capacity of the intestinal mucosa.
4. Inadequate dietary intake of Vitamin B_{12}.

These factors were considered in the differential diagnosis of our patient's anemia. Her stools showed normal fat content, and there was no diarrhea—sprue and idiopathic steatorrhea were ruled out. She had no history of an abdominal operative procedure, nor were there radiological signs of intestinal stasis related to intestinal strictures or blind loops of bowel. Microscopic examination of the stools excluded the possibility of parasitic infestation. Occasionally megaloblastic anemia has been reported in association with the ingestion of anticonvulsants[13, 14]—digitalis was the only drug being administered in this case. There was no radiological evidence of gastric neoplasm. Clinically, hepatic cirrhosis and hypothyroidism[17], syphilis, tuberculosis, pellagra, Hodgkin's disease and chronic nephritis were also excluded.

In 1953 Schilling[18] reported a method for estimating intestinal absorption of Co^{60} B_{12} by assessing urinary radioactivity after a measured oral dose of the tagged vitamin. The Schilling test, in our patient, indicated her ability to absorb orally administered Vitamin B_{12}. This in conjunction with the history of inadequate diet and the complete hematological response to administered Vitamin B_{12} led to the diagnosis of nutritional macrocytic megaloblastic anemia. There was never any evidence of blood loss nor of the presence of a hemolytic process.

It was our intention originally to treat this patient with oral Vitamin B_{12} and thus obtain indisputable evidence of an oral deficiency of the vitamin. However, her precarious clinical state at the time of diagnosis prevented this therapeutic course. It would also have been interesting to have observed the effect of the patient's gastric juice on the Vitamin B_{12} absorption of a proven case of classical pernicious anemia.

Although our patient had developed her anemia over an observation period of nine months, it is likely that her diet was deficient for a much longer period. This feature of the case is in common with other reports[11, 12, 19] stating that three to eight years of deficiency are necessary before megaloblastic anemia and other clinical signs become manifest. The distingushing feature of this case is the presence of persistent histamine fast achlorhydria, rechecked on three occasions after the initiation of therapy. This is in contrast to most of the previously reported cases of nutritional macrocytic anemia.

Summary

A case of nutritional macrocytic megaloblastic anemia in an 85 year old female with persistent histamine fast achlorhydria has been reported. To our knowledge, this is the second account of a case with these clinical features.

References

1. Wills, L. et al: Studies in Pernicious Anemia of Pregnancy. Indian Jour. Med. Res. 17: 777, 1930. Ibid. 20: 391, 1932.
2. Wills, L. and Evans, B. D. F.: Tropical Macrocytic Anemia: Its Relation to Pernicious Anemia. Lancet, 2: 416, 1938.
3. Napier, L. E., et al: Tropical Macrocytic Anemia. Indian Med. Gaz., 73: 385, 1938.
4. Kern, R. A.: Diet as a Factor in the Etiology of Anemia. Ann. Int. Med., 5: 729, 1931.
5. O'Hara, D. and Grewall, J. S.: Unusual Case of Pernicious Anemia. Boston M. and S.J., 197: 129, 1927.
6. Ungley, C. C. and James, G. V.: Effect of Yeast and Wheat Embryo in Anemias: Marmite, Yestamin and Bemax in Megalocytic and Nutritional Hypochromic Anemias. Quart. J. Med., 2: 381, 1933.
7. Groen, J. and Snapper, I.: Dietary Deficiency as a Cause of Macrocytic Anemia. Am. J. M. Sci., 193: 633, 1937.
8. Alsted, Gunnar: Exogenous Pernicious Anemia. Am. J. M. Sci.: 197, 741, 1939.
9. Townsend, S. R. and Begor, F. B: Pernicious Anemia due to deficiency of Extrinsic Factor. Canad. M. Ass. J., 47, 352, 1942.
10. Harrison, R. J., Booth, C. C. and Mollin, D. L.: Vitamin B-12 Deficiency due to Defective Diet. Lancet, 1: 727, 1956.
11. Pollycove, Myron: Apt. L., and Colbert, M. J.: Pernicious Anemia due to Dietary Deficiency of Vitamin B-12; N. England J. M., 255, 164, 1956.
12. Halsted, James A. et al: Mechanisms Involved in the Development of Vitamin B-12 Deficiency. Gastroenterology, 30: 1, 1956.
13. Newman, M. J. D. and Sumner, D. W.: Megaloblastic Anemia Following Use of Primadone. Blood, 12: 183, 1957.
14. Christenson, W. N., Ultmann, J. E. and Roseman, D. M.: Megaloblastic Anemia During Primadone Therapy. J.A.-M.A., 163: 940, 1957.
15. Goldhamer, S. M.: Macrocytic Anemia in Cancer of the Stomach. Am. J. M. Sc., 195: 17, 1938.
16. Strauss, A. A., Meyer, J. and Bloom, A.: Gastric Polyposis. Am. J. M. Sc., 176: 681, 1928.
17. Wintrobe, M.: Clinical Hermatology. 519, 522. Lea and Febriger, New York, 1956.
18. Schilling, R. F.: Intrinsic Factor Studies II. The Effect of Gastric Juice on the Urinary Excretion of Radioactivity after the Oral Administration of Radioactive Vitamin B-12. J. Laborate Clin. M., 42, 860, 1953.
19. MacDonald, R. M., Ingelfinger, F. J. and Belding, H. W.: Late Effects of Total Gastrectomy in Man. N. Eng. J.M., 237: 887, 1947.

Case Report
Myasthenia Gravis
Developing Subsequent to Thymectomy

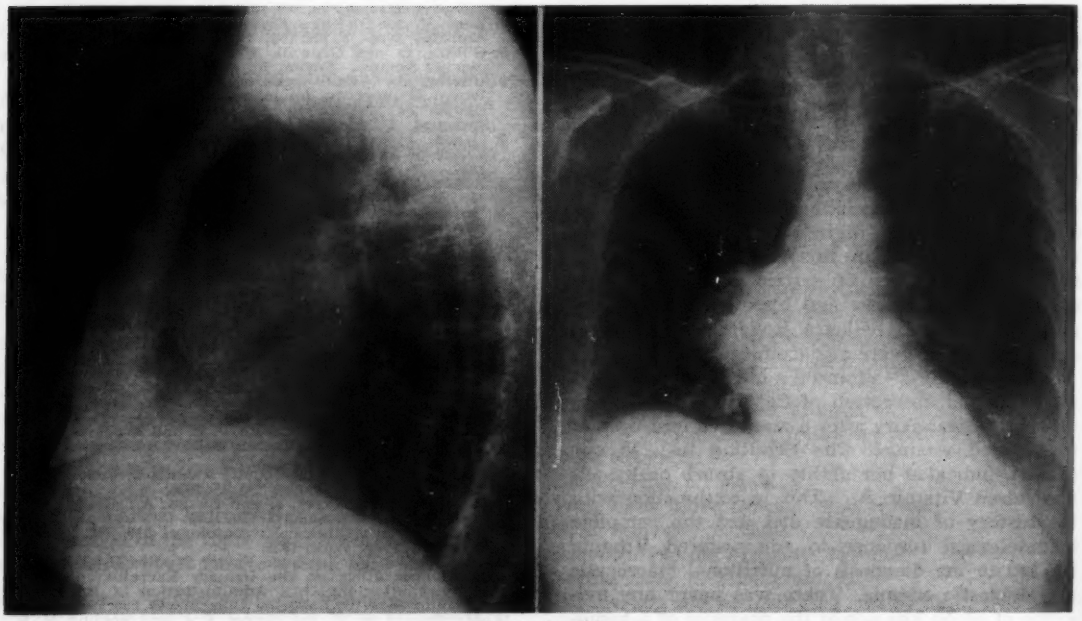
Stephen Szirom, M.D. and Sydney Israels, M.D.

Myasthenia gravis is a chronic disease characterized by weakness following the use of striated muscle. Although the association of myasthenia gravis and diseases of the thymus is well recognized, the pathogenesis of the disease is still obscure. About half of the cases have hyperplasia of the thymus with prominent germinal centres; 15% have thymoma; 10% have enlarged thymus with normal histological appearance[1]. The connection with the thymus is further indicated by the effect of thymectomy on the clinical course of myasthenia. Ever since Weigert in 1901 reported a case of myasthenia gravis with mediastinal tumor, several investigators have tried to establish the true nature of the relationship between the thymus and myasthenia. The hypothesis that the thymus may elaborate some substance which affects the neuromuscular transmission, has gained some supportive evidence. It was found that thymus extracts depress the contraction in muscle-nerve preparations, though they did not produce all the characteristic features of myasthenia gravis[1].

Under the influence of these considerations, after 1939, therapeutic removal of the thymus has been carried out on a large number of cases. In some instances, this resulted in dramatic improvement, and in others the disease was reported to be unaffected or made worse by thymectomy. The severity of the disease and the effect of thymectomy are influenced by the changes in the thymus, the sex and age of the patient at onset and the duration of symptoms[2, 3]. The appearance of the myasthenia gravis after the removal of thymoma is rare. The interval before the onset of symptoms varies from the immediate operative or post-operative state to five years after thymectomy. Our case represents one of these few cases, when symptoms appeared abruptly following thymectomy.

Case History

The case being reported was a 65 year old white female who had been strong all her life and used to hard physical work. In 1931 she noted numbness of the hands with loss of temperature sensation, weakness of arms, fatigue and some chest pain. The symptoms were worse early in the morning, and she had difficulty in getting out of bed. However, she was not entirely incapacitated and was able to continue her work as a hairdresser. In 1935, after taking some unknown liquid medication, marked improvement took place and she remained well for years. However, she has complained of progressively increasing dyspnea since 1935. Sev-



Figures 1 and 2
X-ray of Chest taken pre-operatively indicating Thymoma in Right Anterior Mediastinum

eral years ago she had ankle edema which was attributed to heart failure. In February, 1958 she was admitted to hospital for biopsy of enlarged inguinal lymph nodes which showed histologically a granulomatous reaction. Chest x-ray at that time revealed a circumscribed homogeneous density in the right anterior mediastinum—7 cm. in diameter (Figures 1, 2). A thoracotomy was done and the lesion proved to be a thymoma of the small round cell type weighing 134 gms. Her dyspnea improved after the operation, and she remained well for two months, when weakness of the lips, face and jaw came on abruptly. She had difficulty in swallowing, had transient weakness of the arms and again became severely dyspneic. She could not walk more than 10 steps without resting and began to have short episodes of diplopia. She was readmitted to hospital as a case of cerebral thrombosis although there were none of the usual neurological signs. Subsequently she was transferred to the Winnipeg Municipal Hospitals for long term care.

On examination she was alert, but her face was expressionless, speech was slurred and faded away at the end of each sentence. There was some ptosis, the jaw hung open and she had difficulty in swallowing and breathing. Vital capacity which was recorded on several occasions, ranged between 600 and 800 cc's. Aside from the muscle weakness which was also evident in the neck and respiratory muscles, neurological examination was normal. After eight days of observation, during which the signs and symptoms were constant (with no early morning improvement), 0.5 mgs. of Prostigmine was injected intramuscularly with only subjective improvement on the first dose, but dramatic improvement with the same dose on the next day. The test was repeated on two consecutive days with the same results. The vital capacity rose from 800 cc's to 1300 cc's. No orthopnea or ptosis was noted and mastication as well as swallowing became quite easy. Administration of a placebo failed to provide any objective clinical effect. Neostigmine was started by mouth and she was maintained reasonably free of symptoms on a total daily dosage of 45 mgs. She was discharged from hospital two weeks later, at which time she was able to take solid food and could walk without difficulty; she had no dyspnoea or ptosis and most of her admission complaints had disappeared. Sixteen months later patient still has no evidence of ptosis, walks half a mile without difficulty and she does all her own housework. Vital capacity is 1800 cc's. She continues to remain symptom free on 45 mgs. of Neostigmine per day.

Discussion

There are less than 20 reported cases in the surveyed literature developing myasthenia gravis after x-ray irradiation[4] or surgical removal of thymus. In the case of Fisher and Child[5] respiratory difficulty developed during operation and patient died four days later. Fershtand and Shaw[6] reported a case with a thymic tumor causing chest pain, dyspnea and superior vena caval obstruction, who developed symptoms post-operatively and died 15 months later. In both cases, incompletely removed malignant thymomas were complicated by post-operative myasthenia gravis. B. B. Roe[7] published a case in which myasthenia developed six weeks after total thymectomy and radical mediastinal block dissection. A 33 year old woman was reported by M. G. Madonick[8] who developed myasthenia gravis 15 months following removal of asymptomatic thymoma discovered on routine chest x-ray. Re-exploration of mediastinum revealed residual thymic tissue, but removal of the persistent thymus did not result in amelioration of symptoms. One case reported by T. Ehrenreich[9], six others mentioned in a survey published by L. P. Rowland et al[10] (no detailed report on three cases) four cases seen by K. E. Ossermann[11] (no data) and finally two cases presented in the French literature by P. Veran et al (1956) and A. Meyer et al (1957) bring the total number of cases of which we are aware up to 17.

These case reports are seemingly paradoxical. Appearance of myasthenic symptoms in a previously symptom free patient after thymectomy cannot be caused by the presence of thymic tissue. However, it does not exclude the possibility of thymic influence. Two cases developed symptoms during or immediately after surgery. This may be attributed to the administration of curare, which precipitates the myasthenic crisis, and to the effect of cyclopropane and Demerol, which may have contributed to the symptoms[5]. Also in those cases, where autopsy or re-exploration of the mediastinum was performed months or years after thymectomy, there was always some residual tumor or hyperplastic thymus tissue found. Even with radical mediastinal block dissection, there is the possibility of ectopic gland tissue located in and below the thyroid gland and behind the sternomastoid muscle[12]. Residual thymus or tumor tissue resulting in recurrence of the tumor does not always show up on x-ray, and therefore a negative x-ray does not exclude the possibility of existing thymic tissue.

Those patients who had esophagrams previous to surgery, had x-ray evidence of impairment of the swallowing mechanism, without symptomatic dysphagia[10]. This fact suggests the possibility of "subclinical" pre-operative myasthenia gravis. Hypothetically, it is possible, that there is a delay in the clinical manifestation of the disease and thymectomy fails to interrupt the already initiated sequence of events[7].

Our case had no clinical evidence of myasthenia gravis until seven weeks post-operatively. Symptoms which appeared during the years 1931-35 were not characteristic of myasthenia gravis (worse early in the morning, sensory changes), though subjective sensory disturbances may precede the

first manifestation of the muscular weakness[11]. Further indication against pre-existing myasthenia is the symptomless period of 25 years, which is unusually long for a spontaneous remission, but it is not impossible, to have remission up to 29 years[1].

Also worthy of mention is the uneventful operative and post-operative course of the patient, the abrupt appearance of the muscular weakness, and the relatively low (45 mgs./day) Neostigmine requirement to control the symptoms. The patient feels well and has no signs of myasthenia two years following thymectomy. Survey of 10 case reports by L. Rowland[10] and co-workers indicated that seven of the 10 cases died within two years following the appearance of myasthenia gravis.

There is no satisfactory explanation in this case for the post-operative presentation of myasthenia. The drugs used during surgery are very unlikely to have caused symptoms seven weeks later. X-ray of the chest taken three months and 22 months following thymectomy did not indicate mediastinal pathological findings to suggest recurrence of tumor, though, as it has been stated, x-ray is of limited value. The possibility that both the histologic changes in the thymus and myasthenia gravis may result of a common unknown etiologic factor[13] best explains why removal of the thymus did not prevent appearance of the post-operative symptoms.

Summary

A case report of a 65 year old female patient is presented, in whom myasthenia gravis appeared abruptly seven weeks following thymectomy. The mechanism responsible is obscure. Good response was obtained with Neostigmine and no myasthenic symptoms are evident two years following thymectomy.

Acknowledgment

We wish to thank Dr. S. C. Hershfield for allowing us to use his patient to present this case history.

References

1. Synopsis on Myasthenia Gravis. Am. Journ. of Medicine. 19: 655-742, 1955
2. Ross, R. T.: Thymectomy in the Treatment of Myasthenia Gravis. Lancet. 1: 785, 1952.
3. Simpson, J. A.: An Evaluation of Thymectomy in Myasthenia Gravis. Brain. 81: 112-144, 1958.
4. Eaton, L. M. et al: Thymectomy in Treatment of Myasthenia Gravis. Arch. of Neurol. and Psychiatr. 61: 467, 1949.
5. Fisher, J. H. and Child, C. G.: Myasthenia Gravis Developing Acutely after Partial Removal of a Thymoma. New England J. Med. 252: 891, 1955.
6. Fershtand, G. B. and Shaw, R. R.: Malignant Tumor of the Thymus Gland; Myasthenia Gravis Developing after Removal. Ann. Int. Med. 34: 1025, 1951.
7. Roe, B. B.: Myasthenia Gravis Secondary to Thymic Neoplasm. Journ. of Thoracic Surg. 33: 770, 1957.
8. Madonick, M. G. et al: Myasthenia Gravis Developing Fifteen Months after Removal of Thymoma. Arch. of Int. Medicine. 99: 151, 1957.
9. Ehrenreich, T. and Allen, A. C.: Myasthenia Gravis Following Extirpation of an Asymptomatic Thymoma. Cancer. 11: 173, 1958.
10. Rowland, L. P., Aranow, H. Jr. and Hoefer, P. F. A.: Myasthenia Gravis Appearing after the Removal of Thymoma. Neurology. 7: 584, 1957.
11. Osserman, K. E.: Myasthenia Gravis. 1st ed. Grune and Stratton. 1958.
12. Castleman, B. and Norris, E. H.: The Pathology of the Thymus in Myasthenia Gravis. Medicine. 28: 27, 1949.
13. Eaton, L. M., Clagett, O. T. and Bastron, J. A.: The Thymus and its Relationship to Diseases of the Nervous System. Study of 374 cases. A. Res. Nerv. and Ment. Dis. Proc. 32: 107, 1953.

Mycosis Fungoides

Report on Four Autopsied Cases

Stephen Szirom, M.D. and A. C. Wallace, M.D.

Mycosis fungoides, a descriptive title suggested by Alibert in 1832 emphasizing the clinical appearance, is a disease of the reticulo-endothelial system. Although the skin is primarily affected with the production of fungating, necrotizing tumors, yet any organ may be involved by lesions similar in type histologically to those of the skin. The etiology is unknown. Many suspect some infective agent, but the majority believe that mycosis fungoides is neoplastic in origin. The disease presents considerable diagnostic difficulties. In the early stages, which may last for 15 to 20 years, the cutaneous lesions may present the appearance of a benign rash. Often skin biopsy fails to provide the correct diagnosis at this stage. In the late stage of the disease, terminal transition to lymphatic leukemia, Hodgkin's disease or lymphosarcoma may occur. Some of the lymphomas may manifest cutaneous tumors, clinically indistinguishable from mycosis fungoides.

Clinically, the disease may be classified into three stages: the eczematoid or premycotic, the indurative or plaque stage and the tumor stage[1]. All these stages may present simultaneously or appear independently without progressing through the preliminary stages. The course is prolonged and almost invariably ends fatally. The first stage, resembling psoriasis, exfoliative dermatitis, seborrhoeic dermatitis, lichen planus, eczema or similar banal dermatoses, is usually protracted. Whether these conditions are, in fact, banal dermatoses at this stage, and only later, in certain individuals, develop into mycosis fungoides, or whether that disease is present from the appearance of the first symptoms, is an unanswered question. The latter possibility is predominantly favored. The second stage presents circinate elevated plaques and is of relatively short duration—an average of one or two years. The final stage, developing from a few months to more than 20 years after the appearance of the first symptoms, is characterized by cutaneous tumors. The tumors may vary in size from several millimeters up to 6 to 8 centimeters in diameter, bluish or brownish red in color, bizarre in shape,

elevated sharply from the surface and usually centrally depressed and ulcerated. The life expectancy, by the time this third stage has been reached, is usually short, averaging about six months. Exceptions, of course, are reported—two patients with localized single tumors remained symptomless for years after surgical removal of the tumor[2].

Case Histories

Case I — A 33 year old white male was admitted to the Winnipeg Municipal Hospitals in November, 1952, with the following history: In 1930, he fell off his bicycle striking his right hip. No injury was apparent at the time of the accident, and he was able to carry on with normal activities. Four months later his right hip became painful. A diagnosis of "dislocation" was made, and he was treated in a plaster spica for two months. In 1931 an operation was performed on the right hip. The wound was discharging pus freely post-operatively and this continued until 1932 when another operation was performed with removal of the head and the neck of the right femur. The patient was told that he had a tuberculous hip. Unfortunately, the pathological reports of these operations are not available. The patient was able to work for many years despite multiple discharging sinuses in the area. During 1945, he began to have swelling of the right leg which ultimately developed into elephantiasis. In 1951, the patient was admitted to the CTC and was treated with Streptomycin and PAS. On admission to the Winnipeg Municipal Hospitals in 1952, the patient stated that his health had been good at all times except for his hip. On admission, there were a number of discharging sinuses surrounded by inflamed and indurated areas of skin with elephantiasis of the entire right leg. The remainder of the physical examination was negative. The course in hospital was of gradual but progressive deterioration with breakdown of the area over the right hip to form a foul, ulcerated, necroted mass undermining the skin edges (Fig. 1). At no time were acid fast bacteria isolated from the area by direct smear, culture or guinea pig inoculation. His Mantoux test was positive. A biopsy from the lesion was interpreted as mycosis fungoides. Urethane was given by mouth, with no apparent effect on the lesions. Terminally, thrombosis of the right iliac and femoral veins developed, and he died suddenly.

At autopsy, the ulcerating area about the right hip extended into the subcutaneous tissue, destroying almost all of the right half of the pelvis and the proximal part of the right femur. This tumor-like mass extended along the femoral vessels and into the abdominal cavity. The right femoral and iliac veins contained pale, firm thrombus. The liver was markedly enlarged, weighing 2,300 grams and the spleen was likewise enlarged, weighing 700 grams. Para aortic and para tracheal lymph nodes were firm. On microscopic examination, the skin

Figure 1
Large tumor of the right hip in case 1.

lesions showed squamous epithelium with extensive ulceration and granulomatous changes in the corium characterized by marked pleomorphism and the presence of numerous histiocytes and reticulum cells. Several atypical Sternberg cells were present but eosinophils and mitotic figures were rare. There was patchy necrosis and fibrosis. Similar changes were found in the para-aortic and para-tracheal lymph nodes (Fig. 2). In the spleen, the follicular pattern had disappeared. There were numerous necrotic areas and hemorrhages and some hyperplasia of the medulla but without the typical changes found in the lymph nodes. The liver showed marked fatty metamorphosis.

Case II was a 49 year old white male who was admitted to the Winnipeg Municipal Hospitals in December, 1956. Fifteen years previously, the patient had an erythematous rash on the inner aspect of his right elbow. This rash was not persistent, but came and went. Six months prior to his present admission, he developed a sore about ½ inch in daimeter below his right knee. This area had an erythematous border and a thick crust. The lesion progressed rapidly in size. Multiple similar lesions made their appearance on other parts of his body. The patient was hospitalized and skin biopsy revealed mycosis fungoides. He was transferred to

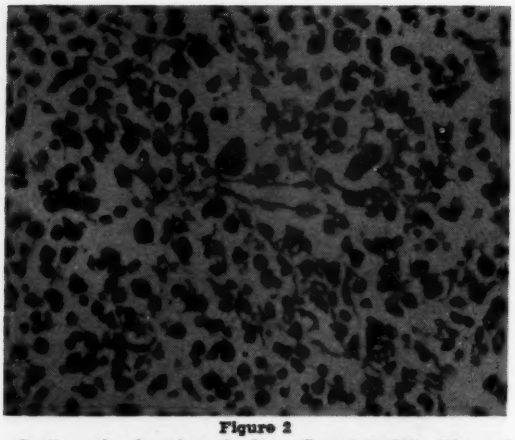


Figure 2

Section of a lymph node from Case 1 (x 500), taken at autopsy. The normal nodal architecture is destroyed and replaced by a pleomorphic infiltrate of lymphocytes, eosinophils, reticulum cells and atypical giant cells resembling Sternberg-Reed cells.

the Winnipeg Municipal Hospitals where, on admission, his body was found to be covered with isolated lesions of various sizes and types. Some of these were maculo-papular and measured only one centimeter in diameter, while others were infected and measured 5 to 6 centimeters in diameter. The remainder of the physical examination was negative. His condition deteriorated from the time of his admission, with progressive anorexia and weight loss. He died of bronchopneumonia 48 days after admission.

At autopsy the skin was extensively involved by large ulcerated areas. No visceral or lymph node involvement was found. There was acute diffuse bronchopneumonia. Microscopically, typical lesions of the tumor stage of mycosis fungoides were found in the skin. (Figure 3):

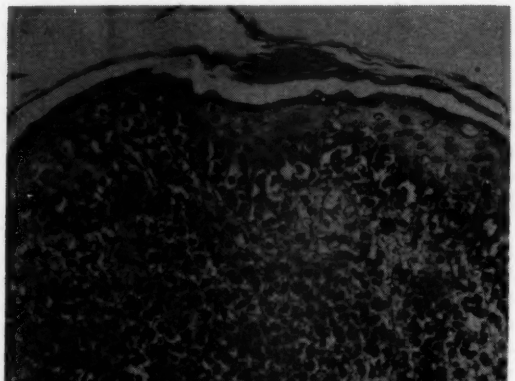


Figure 3

Section of skin (x 225) from Case 2 taken at autopsy. Typical cutaneous lesions of the infiltrative stage are present. The upper dermis is invaded by dense collections of lymphocytes, plasma cells and large, atypical mononuclear cells. The characteristic microabscesses of Darier-Pautrier are present in the epidermis.

Case III — This 60 year old white male was admitted to the Winnipeg Municipal Hospitals in December, 1957 with a history of skin rash since 1950. This rash started on his nose as a small raised area the size of a dime. The lesion would come and go. The rash, however, gradually spread to other portions of his body and consisted of erythematous, scaling lesions which caused him very little discomfort. In 1956, the lesions started to become larger, some of them began to break down and they were quite itchy. Examination at that time revealed erosions of the right side of the nose, involvement of the right eyelid by granulation tissue and innumerable lesions over the entire body (Figure 4). Some had the appearance of the plaque

Figure 4

Lesions of the infiltrative stage with extensive ulceration of the nose and left orbit — Case 3.

stage. Although biospy at this time was not diagnostic, it was felt this was probably due to the pre-tumor phase of mycosis fungoides. He was given a course of nitrogen mustard, with improvement. In October, 1957, he had an acute exacerbation of the disease. Nitrogen mustard was ineffective and spray radiation produced little or no improvement. Leukopenia precluded the use of other cytotoxic agents. All the lesions over his body became necrotic and grossly infected. He died within a few weeks of his transfer to the Winnipeg Municipal Hospitals.

At autopsy, the entire body was covered with pink, orange and silvery lesions. The lesions were confluent over the face, abdomen, groins, back and neck. The nose had completely disappeared, the ethmoid bones being exposed. Although both orbital areas were eroded, the eyes were intact. Microscopic examination revealed pleomorphic infiltration in the dermis with some atypical giant cells and "micro abscesses" compatible with mycosis fungoides. No other organ involvement was found. Death was due to extensive bilateral bronchopneumonia and pulmonary infarction.

Case IV was a 45 year old white female. Her skin lesions started in 1948 with the development of raised patches on her back and abdomen. The lesions were very itchy. She received ultra violet treatment, with some symptomatic improvement. Using applications of various lotions, she got along fairly well until 1956 when her symptoms became worse. At this time a biopsy was taken and a diagnosis of mycosis fungoides was made. X-ray therapy, in combination with cortisone, was started. The x-ray treatment was continued at varying intervals for about a year and resulted in considerable initial improvement. During the summer of 1957, her skin appeared fairly normal except for some radiation dermatitis. By late 1957, however, the radiation therapy lost its effectiveness in the alleviation of symptoms. The tumor stage of the disease developed early in 1958 and many of the lesions showed necrotic changes at this time. She received intravenous nitrogen mustard therapy, with equivocal results. Later, steroid therapy was resumed and appeared to be of benefit. Temporary regression of the lesions was obtained by the intravenous and oral use of Cyclophosphamide (Figures 5 and 6). Despite this improvement her course was that of rapid deterioration. She developed severe bronchopneumonia and died in January, 1959.

At autopsy, huge nodules with ulceration were present on the face, chest, scalp, upper arms, axilla, trunk and thighs. Multiple pulmonary abscesses were found. Enlargement of axillary, femoral, mediastinal and hilar nodes was present. Microscopically, the skin showed infiltration with pleomorphic cells. lymphocytes and large monocytes. Some giant cells, resembling the Reed-Sternberg type, were seen. The lymph nodes presented an appearance similar microscopically to that seen in the skin.

Discussion

The age (33, 45, 49, 63) and sex (three males, one female) distribution of the cases in this report coincides with the opinion expressed in the literature that mycosis fungoides is more common among men and usually arises in the middle or later adult life. The duration of the disease ranged from seven to 20 years from onset to death. Localized rashes and sores were the early manifestations of the disease with generalized cutaneous involvement developing later. The diagnosis of mycosis fungoides

Figure 5

Typical cutaneous lesions of the tumor stage, from Case 4 immediately prior to therapy with cyclophosphamide.

Figure 6

Same case as Figure 5 after a 12 day course of cyclophosphamide.

was not suggested until the plaque or tumor stage had appeared. Once the tumor stage is reached, necrosis of the growths occurs and secondary infection of the lesions is inevitable. In our four cases the suppurative involvement, resistant to all treatment, was prominent. Although eosinophilia has been reported in this condition[1, 13] in our cases, laboratory tests yielded no specific information.

It is a widely prevalent impression that a high percentage of mycosis fungoides cases terminate with visceral involvement or evidence of leukemia, Hodgkin's disease or lymphosarcoma[4, 9, 10]. Some writers, however, have emphasized the possible fallacies in evaluation of this, notably the danger of confusing dermatopathic lymphadenitis with lymphatic involvement by the neoplasm, and of labelling a case of lymphoma or leukemia as mycosis fungoides merely because of cutaneous involvement[1, 6, 8]. Two of our four cases, 1, 4 showed lymph node involvement, and none showed general visceral involvement. Case I is certainly an atypical one, especially in its skin distribution; only one of the three "typical" cases showed lymphoid involvement. Bone marrow invasion was absent in all four cases; this is in accord with other experience[4, 7, 11].

Antimitotic agents are used in therapy of mycosis fungoides but the results of treatment are discouraging. In this report, one case was treated with Urethane alone and the remaining three received combinations of radiation therapy, nitrogen mustard or its derivatives and steroids. Good temporary effects were observed with radiation therapy, but the lesions soon became radio resistant.

Summary

The clinical and pathological features of four cases of mycosis fungoides seen at the Winnipeg Municipal Hospitals during the past ten years have been presented.

We wish to thank Dr. A. J. W. Alcock, Medical Director, Winnipeg Municipal Hospitals, for supplying the photographic illustrations as well as his assistance in preparing this paper.

References

1. A. C. Allen: The Skin, First ed. C. V. Mosby Comp., St. Louis, 1954.
2. J. T. Ingram: Mycosis Fungoides. The Practitioner. 177, 170-175, 1956.
3. P. E. Hammerberg and P. Bastrup-Madsen: Treatment of Mycosis Fungoides with Nitrogen Mustard. Acta Med. Scand. 151: (4), 1955. pp. 317-320.
4. J. E. LeDonne: Mycosis Fungoides. Report of a case terminating in Myclogenous Leukemia. The New England Journal of Medicine. 250: (6), 240-242, 1954.
5. S. M. Bluetarb: Is Mycosis Fungoides an Entity? Arch. of Dermatology. 71: (3),293-302, 1955.
6. W. F. Lever: Histopathology of the skin. Second Ed., B. Lippincott Co., Philadelphia and Montreal, 1954.
7. A. Poulsen: On Mycosis Fungoides. Acta Dermatol-venereal. 21: 365, 1940.
8. O. Ormsby and C. W. Finnerud: Mycosis Fungoides. Arch. Dermat. Syph. 27: 631, 1933.
9. E. D. Cawley, A. C. Curtis and F. E. K. Leach: Is Mycosis Fungoides a Reticuloendothelial Neoplastic Entity? Arch. Dermat. and Syph. 64: 255, 1941.
10. H. F. Heite and P. Socha: Hautigkeitsanalytische Untersuchungen Zur Symptomatologie Der Mycosis Fungoides. Arch. F. Dermat. U. Syph. 193: 118, 1951.
11. R. F. Tilley and D. C. Smith: Bone Marrow Changes in Mycosis Fungoides. Journal Invest. Dermat. 14: 387, 1950.
12. F. R. Hasericke, J. H. Richardson and D. J. Grant. Remissions of Lesions in Mycosis Fungoides following Topical Application of Nitrogen Mustard. Cleveland Clinic Quart. 3: (144), 1959.
13. W. K. Wills and G. Hadfield: Mycosis Fungoides, Brit. J. Dermat. 37: 113, 1925.

SNELL'S DRUG STORES

ACADEMY ROAD AT QUEENSTON STREET
HU 9-1155 HU 9-5989

GRANT AVENUE AT NIAGARA STREET
GL 3-6239 (two phones)

PHARMACISTS
L. F. Munroe C. G. Brigden
R. A. Duff D. J. Berryhill
G. H. Edmonds

HOURS
9 A.M. to 10 P.M. Weekdays
Noon to 10 P.M. Sundays
Outside above hours please call HU 9-2962

THE ANNUAL MEETING OF THE
MANITOBA MEDICAL ASSOCIATION
(Canadian Medical Association, Manitoba Division)
will be held on
SEPTEMBER 26th and 27th, 1960
MARLBOROUGH HOTEL, WINNIPEG

...they need your help more than ever

Ortho Kit*

WHENEVER A DIAPHRAGM IS INDICATED

Ortho

*Trade Mark

Contains Ortho-Gynol* . . . the most widely prescribed vaginal jelly . . . and an Ortho* Coil Spring or Flat Spring Diaphragm of the size you prescribe.

1468

"It is not so much the great theoreticians upon whom the health of the community depends, as the huge army of family doctors who succour the ailing from hour to hour."[1]

For tiny mishaps that loom so large, the doctor can both relieve pain . . . and provide antisepsis . . . through

Bradosol Cream

with NUPERCAINE

Bradosol® (domiphen bromide) 0.05% with Nupercaine® (cinchocaine B.P.) 0.5%, in a water-washable base; tubes of 1 oz.

1. Sigerist, H.E.: The Great Doctors. New York, Doubleday & Company, Inc., 1958

CIBA
DORVAL • QUEBEC

Editorial

S. Vaisrub, M.D., M.R.C.P. (Lond.), F.R.C.P. (C.), F.A.C.P., Editor

Municipal Hospital—Citadel of Geriatrics

Old actors, by their own admission, are as fond of applause and as apprehensive of stage-fright as are the young amateurs. Old sea dogs, by their own confession, are as happy when the twinkling lights of the gay seaport are in sight, and as sick when the sea is choppy, as are the young tars. Old politicians, their testimony notwithstanding, are as given to making rash promises with great gusto, and breaking them with equal unconcern as are the young novices. Old editors — it is whispered in strict confidence — are as faithful to the time worn cliché and the pompous pronouncement, and as fearful of the approaching deadline as are their young confreres. Wherein, then, lies the difference between the old and the young?

It may be argued, of course, that as long as actors act, sailors sail, politicians politic and editors edit, they are not truly old, but merely "seasoned." They are experienced, toughened, tested by time — old only in the affectionate sense of the word. Theirs is not the old of old age, but the old, of "old soldiers (who) never die" and "old friends (who) are best".

When does, then, true old age begin? Does it commence with retirement at a chronological age set arbitrarily by the employing powers, be they military, civil, business or academic? Or is old age individualized — a matter of complex criteria, stigmata and parameters? Is a man old when his teeth decay, his hair is grey, his libido at bay, or are the signs of senility more subtle? Perhaps, an overpolite "sir" from a previously disrespectful youngster, an unasked for assistance to a coat in a cloak room, an unsolicited offer of a seat by a pregnant woman in a crowded bus, a respectful silence by bored listeners in lieu of a deserved rebuttal to an uttered absurdity — all variants of the deferential and solicitous attitude of the erstwhile pugnacious world are the augurs of the dreaded "molestas senectutas".

Many, however, would deny validity to either physical criteria — dentures, wrinkled epidermis, arcus senilis, shuffling gait, or environmental attitudes—be they reverential or scornful. They would take their cue from the proverb "you can't teach an old dog new tricks". A man, according to their view, is old when he is no longer capable of learning, when his mind is no longer open to new ideas. Was not Michaelangelo 80 years young when he turned to architecture? Was not Grandma Moses a mere youngster of 78 when she first took up painting? Was not Bertrand Russell in his youthful 70's when he wrote his first novel? Sensitivity more than sensuality, creativity rather than procreativity are — they contend — the determinants of youth.

It can be readily seen that there are no unanimously agreed upon criteria of senility. Nor are there reliable subjective guides. Rarely does the aging male of the species, and hardly ever the female admit to being old. Leaning heavily on the adage "you are as old as you feel," they shuffle merrily along, confident that they feel young, and content, now that they are no longer capable of setting bad examples, to give good advice. Small wonder, then, that as an office specialty Geriatrics does not prosper. How can it, indeed, if no oldster would admit to being old?

The failure of geriatrics to flourish as an office specialty does not imply, however, that it is not a specialty at all, or that it is not practised. Many physicians devote a great deal, if not all, of their time to the study of Gerontology and the care of the aged. They do it largely in hospitals, which are dedicated primarily to the care of the chronically ill older patients. In these specialized hospitals they find the trained personnel and the facilities required for adequate treatment of their geriatric patients.

That the older patient requires specially trained personnel, a special approach and, indeed, a special philosophy of treatment is self-evident, all the facetious remarks in the preceding paragraphs notwithstanding. The elderly sick present physical, mental and social problems that demand services of trained teams of doctors, nurses, dietitians, physiotherapists, psychologists, speech therapists and social workers. They must not be hurried along in and out of the wards of active general hospitals, nor should they be eased into a vegetative existence in some lonely room. Theirs is a crying need for rehabilitation and understanding.

The Municipal Hospital — our honored guest this month — is primarily a geriatric hospital. With our lengthening life span, and consequent increasing number of the aged sick, this distinguished member of the hospital family is destined to expand and play an ever more important role on the local medical stage.

Ed.

IN DEBILITATING DISEASE

Patients receiving

NILEVAR®

Eat more...
Feel better...
Recover faster

Compared to control patients, those receiving Nilevar (brand of norethandrolone) have repeatedly demonstrated more rapid and more complete recovery from serious acute illness and increased comfort and well-being in chronic illness.

A multitude of case histories are now adding individual clinical color to the earlier controlled investigations which defined the actions of Nilevar as an effective aid in reversing negative nitrogen balance and in building protein tissue.

In typical case reports such gratifying comments as these appear:

Underweight –"Appetite considerably increased within one week. Sense of well-being and vigor increased along with increased appetite."

Prematurity (Birth weight: 2 pounds, 4 ounces) – "Gradual improvement in appetite and capacity for formula. . . . Excellent progress and weight gain for a very immature infant."

Carcinoma of the Uterus –"Within four days appetite became excellent, took full diet. . . . More ambition while on Nilevar. Enjoys life. Takes part in church and other social affairs."

Third Degree Burn–". . . soon began eating all that was offered. . . . Began to show signs of hope for recovery. . . . Perhaps one of the greatest changes was in the appearance of his wounds which were so very much improved."

The dosage for adults is 20 to 30 mg. daily in single courses no longer than three months. For children the daily dosage is 0.5 mg. per kilogram of body weight, in single courses no longer than three months.

Nilevar is supplied in tablets of 10 mg., ampuls of 25 mg. (1 cc.) and Nilevar Drops of 0.25 mg. per drop.

G. D. SEARLE & CO. OF CANADA LTD., 247 QUEEN ST., E., BRAMPTON, ONT.

Book Reviews

A Practical Guide to General Surgical Management: Julian A. Sterling, M.D., Assistant Professor of Surgery, Graduate School of Medicine, University of Pennsylvania. Cloth $3.00, pp. 67, Vantage Press Inc., 120 West 31st Street, New York 1, N.Y., 1959.

This small volume as its name denotes is a guide to surgical management. It was prepared specifically for the use of internes and residents at the Albert Einstein Medical Centre in Philadelphia.

As a general "Guide" it definitely has a place and should be very helpful for whom it was written and for all internes. Many surgeons will not agree with some specific points of surgical procedure and of pre and post operative management. However, the author must of necessity be dogmatic and leaves discussion of the whys and wherefores to other publications.

A number of normal values of commonly requested laboratory tests are included under the section "Miscellany." This section also contains information specific for the A. E. Medical Centre and of not much use to others.

This little book should be of help to surgical internes and residents, but is no better or worse than many others previously published.

M. L.

☆

Heritable Disorders of Connective Tissue: Victor A. McKusick, M.D. Illustrated Second Edition, 1960. The C. V. Mosby Company, St. Louis. pp. 333. Price $12.00.

The first edition of this book appeared in 1956. It was a modest effort based on a series of articles previously published in the journal of Chronic Diseases. The second edition boasts of an expansion by more than one half of its original size. The added 130 pages and 80 new illustrations give the book a touch of newness not usually present in successive editions of medical texts.

The book deals primarily with rare diseases. The Marfan Syndrome, the Ehlers-Danlos Syndrome, Osteogenesis Imperfecta, Pseudoxanthoma Elasticum, the Hurler Syndrome are not conditions frequently encountered in daily practice. It would, therefore, seem at first glance that the book was intended for the dabbler in curios, the lover of the bizarre. This impression is deceptive. The book transcends the narrow confines of the esoteric, for, apart from its extensive clinical studies, it delves deeply into two subjects, which are of interest to every practitioner—the subject of genetics and that of connective tissue.

The author's approach is that of attempted synthesis rather than presentation of scattered information. It is an approach inspired by his conviction that "hereditary syndromes are tools for study of the normal situation, in this case for the elucidation of connective tissue" and his belief which he shares with William Harvey whom he quotes: "Nature is nowhere accustomed more openly to display her secret mysteries than in cases where she shows traces of her workings apart from the beaten path".

S.V.

☆

Drugs of Choice, 1960-1961: Walter Modell, M.D. Editor. Second Edition. The C. V. Mosby Co., St. Louis, 1960. pp. 958. Price $13.50.

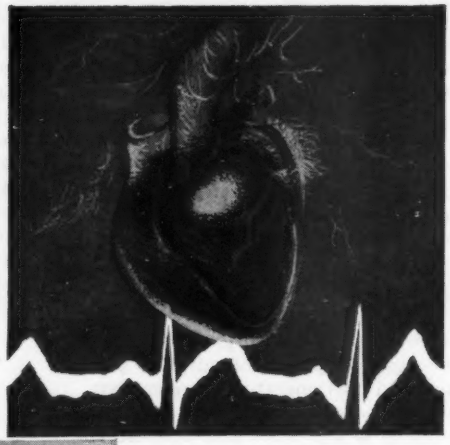
This reviewer always felt that somewhere there must be a book which is a happy medium between the readable bright brochure dedicated to the glorification of a particular drug for a particular clinical condition and a forbidding encyclopedic text book of pharmacology in which a long list of drugs for the self same condition is dealt with in great detail with detached objectivity leaving the reader no guide-posts for selection.

The discovery that such a book was in existence since 1956 came to the reviewer as a pleasant surprise when he was presented with the 1960 edition of "Drugs of Choice". Having missed the first edition and thus saved all his superlatives, he could now apply them without fear of repetition to the second. This, indeed, is a most satisfying book. A joint effort in which 47 authorities participate in a lively appraisal of drugs, it steers skillfully between the Scylla of sticky subjectivity and the Charybdis of total detachment. The authors discuss the basic problem presented by the clinical situations for which the drugs are intended and after a brief outline of the pharmacologic properties of the drugs under consideration, proceed to state their preferences, supporting them with solid clinical and experimental evidence. Although sticking their necks out at times where straddling fences (excuse the mixed metaphor!) would be more advisable, they do on the whole an excellent job of appraisal.

For the benefit of those who have read the first editions it may be mentioned that in the second one, eight chapters have been added, and a number of new authors contributed, thus bringing it up to date.

S.V.

in congestive heart failure



PURODIGIN*
(Crystalline Digitoxin, Wyeth)

Indicated in congestive heart failure, PURODIGIN achieves and maintains digitalization with the smallest oral dose of all cardioactive glycosides. It offers high potency, complete absorption, steady maintenance, uniform action.

Supplied: PURODIGIN TABLETS—0.2 mg.—bottles of 30, 100 and 500; 0.15 mg. and 0.1 mg.—bottles of 100 and 500. PURODIGIN AMPULS—0.2 mg.—boxes of 6.

COMBINED THERAPY
to keep the patient steadily compensated and edema-free

THIOMERIN*
(Mercaptomerin Sodium, Wyeth)

Indicated for diuretic therapy, THIOMERIN produces smooth, effective fluid loss. It may be administered subcutaneously, intramuscularly or intravenously.

Supplied: THIOMERIN SOLUTION — ready for injection — vials of 2 cc. and 10 cc.
THIOMERIN LYOPHILIZED — vials of 10 cc. and 30 cc.

Wyeth
Reg. Trade Mark
WALKERVILLE, ONTARIO

* Reg. Trademark

Questions and Answers Page

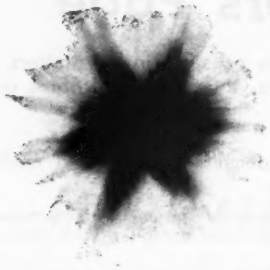
In order to keep the medical profession informed as fully as possible in all matters relating to Association business, medical economics and prepaid medical care, this page welcomes questions pertaining to these fields.

Question: What is the difference between "Service Plans" and "Indemnity-type" prepaid medical care coverage? . . . . M.D.

(This question was a frequent problem at a recent meeting of the medical members of the Manitoba Medical Service. The answer supplied here has been compiled with the aid of the administration of Manitoba Medical Service.)

Answer: The "Service Type" plan outlined here is that of comprehensive coverage as it exists under M.M.S. However, coverage under service plans varies with each plan, and also varies from plan to plan.

	Service	Indemnity
1. Objective	Eliminate financial burdens of illness entirely.	Reduce financial burdens of illness.
2. Operation	Non-profit in operation. No shareholders. Board of Management definite relationship to recognized medical organizations.	Profit in operation. Has shareholders No relationship to any organized medical body.
3. Income limits on contract holders	Reserved usually for low and average income persons.	No income limits.
4. Contract	Between the plan and the subscriber, and also between the plan and the doctor.	Between the insurer and the policyholder only. Assignment may be made to doctor and insurer pay doctor directly.
5. Benefits	Comprehensive and complete usually (although of course may vary. Tend to be broad in scope).	Limited, with deterrent and control features.
6. Doctor's Fees	Fees paid according to schedule designed by organized medical body. Paid by plan to doctor. Usually accepted by doctor as "paid-in-full" for type of service covered. Unguaranteed. Extra billing only under special circumstances. Fee tariff is not part of the contract with the subscriber. Expense he can incur is unlimited.	Schedule of allowances set up by insurance actuaries paid by insurer to the policy holder. Contractual allowances towards doctors usual and customary charges. Extra billing at the discretion of attending physician for other services.
7. Jurisdiction	Boards of Trustees. (Canada - U.S.A.). Provincial legislatures. Divisions of C.M.A. State Medical Societies (U.S.A.).	Superintendents of Insurance (Canada) State Insurance Commissioners (U.S.A.)
8. Examples of plans	Manitoba Medical Service and other C.M.A. division-sponsored plans (Vancouver to Halifax). Blue Cross Plans (Canada - U.S.A.). Blue Shield Plans of U.S.A.	British Pacific Insurance Co. Canadian Premier Life Insurance Co. Great-West Life Assurance Co. Ltd. Mutual of Omaha Occidental Life Insurance Co. etc., etc.



Trichomonad explosion with VAGISEC* liquid and jelly reduces "psycho-social" tension

Vaginal trichomoniasis is recognized as a disease of "psycho-social significance"[1] because the acute symptoms of profuse, scalding leukorrhea, itching and swelling, compounded by painful, difficult coitus[2] often lead to family tensions.

One VAGISEC office treatment brings immediate symptomatic relief. Decker[3] reports immediate relief of symptoms following the first office treatment with VAGISEC liquid and jelly in all 64 acute cases studied.

Cure rates of 93.1% using negative cultures for three consecutive months. Giorlando and Brandt[4] demonstrated repeated negative cultures for three to eight months in 54 of 58 vaginal trichomoniasis patients treated with VAGISEC liquid and jelly. Lack of cooperation was held responsible for the four failures.

VAGISEC explodes trichomonads within 15 seconds of contact. VAGISEC's wetting, detergent, and chelating agents dissolve the mucus protecting the trichomonads, remove waxes and lipids from the cell's membrane, denature its proteins. The parasite then imbibes water, swells, and explodes.

Prevent recurrence—prescribe RAMSES* for the husband. To prevent re-infection, most physicians advise the use of a prophylactic during coitus for four to nine months following the wife's treatment,[3,5] and when they do, 50.3% specify RAMSES.

References: 1. Trussell, R. E.: New York Med. *13*:717 (Sept. 20) 1957. 2. Karnaky, K. J.: South. M. J. *51*:925 (July) 1958. 3. Decker, A.: New York J. Med. *57*:2237 (July 1) 1957. 4. Giorlando, S. W., and Brandt, M. L.: Am. J. Obst. & Gynec. *76*:666 (Sept.) 1958. 5. Weiner, H. H.: Clin. Med. *5*:25 (Jan.) 1958.

VAGISEC* LIQUID AND JELLY

*Trade-marks

JULIUS SCHMID (Canada) Ltd., 32 Bermondsey Rd., Toronto 16

Social News

Reported by D. M. H. Huggins, M.D.

Our Katy is progressing favourably and reports she will be ready to wear a Bikini when swimming time rolls around. All the best to you, Kay, and we hope to see you around soon.

☆

Mrs. Nona Mari, a city soprano, wife of Dr. Paul Mari, of St. James won the "Rose Bowl" at the recent Musical Festival in Winnipeg. She was also the winner of three other major vocal awards. Congratulations Nona!

☆

Mrs. T. E. Beighton was elected a Regent of the I.O.D.E. Municipal Chapter recently. Wives of the Medical profession are favourites for this position apparently, as Mrs. Beighton succeeded Mrs. Roper Cadham.

Mrs. Roper Cadham recently received an honourary life membership in the I.O.D.E. for her work in the society.

☆

Dr. Max Desmarais who is presently with the World Health Organization has been doing a tremendous piece of rehabilitation work in Morocco following the substitution of surplus aircraft engine oil for pure olive oil for kitchen use. In company with the other Canadians he has been sent to the districts where the need has been the greatest, and reports that about 20% of those afflicted with "Mekness Sickness" will be completely cured, while others are showing a definite improvement. Good International work, Max!

☆

Dr. Don Wright, M.D. '48, sent Kay a cheery note from Aylmer, Ontario, where he is practicing. He describes himself as "An Eastern subscriber, with an everlasting western glance." (The prairies are in your blood Don).

He saw Ken McGavin and Darcy Bruce, class of '45 at the O.M.A. Curling Bonspiel.

☆

Dr. Donald H. Williams, class of '31, who has been a lecturer at the University of British Columbia since 1940, has recently been appointed head of a new department of Continuing Medical Education at U.B.C. Congratulations Don, from all your friends in Manitoba.

☆

Dr. Patrick Redman of Class '56 we hear is practicing in Trinidad. What a grand place to live.

Dr. James F. Lind, a graduate of Queens University, has been named a Markle Fellow. He will do his work at Manitoba in the Department of Surgery. Welcome Dr. Lind!

☆

The Welcome Mat is also out for Dr. Richard A. Lim, who has been a Fellow in Surgery at the Mayo Foundation in Rochester, Minnesota. Dr. Lim is now practising surgery in St. Boniface.

☆

The stork has made calls to several doctors' homes recently:

In Toronto he left twin "bundles of charm" to Dr. and Mrs. Clive Wightman (nee Patricia Pickard). The unexpected twin brothers for Clive arrived on March 30th.

☆

To Dr. and Mrs. Fletcher D. Baragar on March 31st a son, Ian Bruce, a brother for David and Anne.

☆

To Dr. and Mrs. R. S. Butler of Dauphin, a son, Jeffrey Raymond.

☆

To Dr. and Mrs. William F. Campbell on April 22, a daughter, Jane Anne.

☆

To Dr. and Mrs. Stuart Carey of Clearwater Lake Hospital on March 22, a fourth son, Patrick Leonard—a brother for Paul and the twins, Michael and Murray.

☆

To Dr. and Mrs. Marvin Raynes on March 17th, a daughter Naomi Susan.

☆

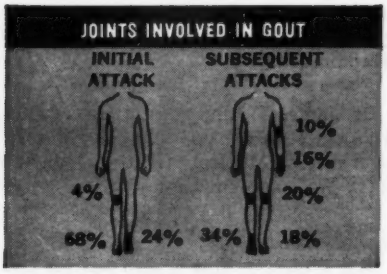
To Dr. and Mrs. Arnold G. Rogers, on March 19, a son.

☆

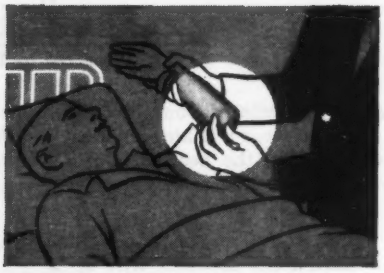
To Dr. and Mrs. M. L. Woods, on March 20th, a daughter, Martha Lee, a sister for John Kai.

☆

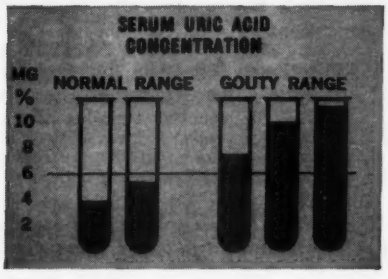
To Dr. and Mrs. John Maclean on April 25th. 1960, a daughter Pamela.



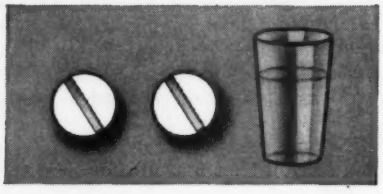
1. Recurrent joint pain followed by long periods of complete remission. *(Percentages refer to incidence.)*



2. Enlargement of bursae such as in this case involving the olecranon bursa.



3. Elevated serum uric acid levels.



4. Colchicine test: full dose (0.5 mg.) every 1 to 2 hours until pain is relieved or nausea, vomiting or diarrhea occur. The test requires usually 8 to 16 doses. Pain relief is highly indicative of gout.

FROM THESE FINDINGS...SUSPECT GOUT:

℞ BENEMID
PROBENECID

A SPECIFIC FOR GOUT

Once findings point to gout, long-term management can be started with BENEMID. This effective uricosuric agent has these unique benefits:

Urinary excretion of uric acid is approximately doubled.
Serum uric acid levels are reduced.
Uric acid deposits (tophi) in tissues are mobilized.
Formation of new tophi can often be prevented.
Fewer attacks and severity is reduced.

RECOMMENDED DOSAGE: 0.25 Gm. (½ tablet) twice daily for one week followed by 1 Gm. (2 tablets) daily in divided doses.

BENEMID is a trade-mark of Merck & Co. Limited.

MSD
Merck Sharp & Dohme
Division of Merck & Co. Limited
Montreal 30, Que.

Association Page

C.M.A. Public Relations Committee Meeting, Toronto April 1st and 2nd, 1960

The meeting was held at C.M.A. House and in the Park Plaza Hotel in Toronto. Representatives from all the Provincial Divisions were present. Dr. Everett F. Crutchlow of Montreal was in the chair.

Considerable interest was shown in the results of the public opinion survey conducted here in Winnipeg last summer by the Manitoba Health Service. The item dealing with the evident satisfaction with both subscription rates and benefits received was accepted with an enthusiasm that became dampened when the items dealing with the attitudes of the public towards the fact of downward pro-rating of doctors' claims became known. The committee was also interested in the misapprehension in the public mind regarding who operates the plan. The British Columbia Division reported that it now has a special committee with representation from the several prepaid medical care plans operating there. The purpose of the committee is to provide fuller information both to the profession and the public because there is concern lest misunderstandings develop, particularly between the profession and the plans, leaving the latter less effective instruments of public service. A resolution was passed on to the C.M.A. Executive that serious consideration be given to increased public relations activity in the field of approved voluntary prepaid medical plans with a view to keeping the best features of these before the profession and the public.

The most interesting Divisional report was that provided by Saskatchewan whose representative gave us considerable insight into the situation there as it presently stands. He indicated that there was very strong support in the profession for the stand taken by the Saskatchewan Association in its dealings with the C.C.F. Government. Each Saskatchewan doctor has been equipped with an information kit so that he has, for his own use, all the material possessed by the Divisional office, with which to refute the C.C.F. claims regarding the proposed legislation. It is also hoped that many Saskatchewan doctors will be able to appear on television for one or two minutes to explain to their patients and others in their communities why they object to a compulsory government controlled scheme. It was made evident that the Saskatchewan Division had already had some success in negotiating with the Provincial Government. The profession had been asked to name several members to an advisory committee to study the proposed government legislation but had insisted that no representatives would be named until the committees' terms of reference had been broadened to in include a study of health needs as a whole as well as the projected medical care plan itself. Also the time limit of a 1961 report from the advisory committee has been dropped. It also appears that the enabling legislation which was to be introduced at the present session of the Saskatchewan Legislative Assembly has been postponed. Dr. Forrester, the Saskatchewan representative, felt therefore that they had made considerable progress, but felt that they needed more support particularly from the Canadian Medical Association.

The Friday evening session consisted of a reception and dinner after which the meeting was addressed by two speakers, Mr. Eamon Park, Assistant National Director of the United Steel Workers of America and Mr. H. L. Hickey, General Public Relations Manager of Massey-Ferguson Ltd. The object was to determine "how others see us." Mr. Hickey spoke last, and since his talk was at least partly designed to show that some segments of the public view the profession with respect and understanding it was a pleasant reprieve after the hammering Mr. Park gave us, but also for that reason produced little discussion. Mr. Park criticized us bluntly and persistently and I believe honestly. He left no question of his belief that the profession was often largely self-seeking and in its relations with labour organizations intransigent and arrogant. He further indicated his belief that certain medical care plans had become excessively costly due to abuse on the part of participating doctors. While one could not agree with what Mr. Park had to say, there was no question in my mind that he meant what he said, and was telling the truth as he believed it to be. Mr. Park was also very frank with us in stating that the only answer to the provision of medical care from his point of view was the complete socialization of medical care. He left no doubt at all of the intention of labour in this regard and no doubt about the single-minded purposefulness with which this object would be pursued.

While it was not a pleasant experience listening to this kind of address, it was evident that Mr. Park had done exactly what he was asked to do when he was invited to speak. He had been asked to tell us how the profession looked to him and he had done so forthrightly and frankly, and for this we owe him our thanks. There was no inclination in the committee to take Mr. Park's remarks

Indicated in Amenorrhea and at the Menopause

OESTRILIN

TABLETS

NATURAL OESTROGENIC HORMONES

PLAIN:

For Oral Estrogenic Therapy
Each tablet contains 0.65 mgm. (6,000 I.U.) of Conjugated Estrogenic Substances

DOSAGE: 1 to 3 tablets daily.

WITH PHENOBARBITAL:

For Oral Estrogenic Therapy with Sedation
Each tablet contains 0.65 mgm. (6,000 I.U.) of Conjugated Estrogenic Substances and Phenobarbital 30 mgm. (½ gr.)

DOSAGE: 1 to 2 tablets daily

Bottles of 25 and 100 tablets

DESBERGERS LIMITED
MONTREAL **CANADA**

lightly. He appeared as the responsible representative of a large and influential organization. If other large and influential bodies of public opinion see the profession in the same light, we can surely expect little sympathy from them in the future. It is more than evident that our public relations have been considerably less than adequate. Two resolutions were passed to the C.M.A. Executive partly, at least, as a result of the discussion of Mr. Park's speech. The one suggested that in order to keep abreast of trends of Canadian thinking in health matters, the C.M.A. and the Divisions should frequently meet with representatives of labour, political parties and organizations such as Chambers of Commerce, etc. for exchange of opinions. The second reads as follows: "That in veew of the problems caused by that minority of the medical profession who allegedly abuse the prepaid plans and/or other forms of insurance and thus do great injury to the public impression of the profession, be it resolved that the Executive of the C.M.A. through its Divisions be asked to contact the several Colleges of Physicians and Surgeons across Canada informing them of the importance of this matter and asking their co-operation in taking all possible corrective steps.

All in all this was not a pleasant meeting although the arrangements were excellent, but a rather uncomfortable and difficult one and certainly a very thought-provoking one.

R. H. McFarlane, M.D., Chairman,
Public Relations Committee.

Obituaries

Dr. Ruth Mathers

Dr. Ruth Mathers (Marion Ruth Pearson) died April 5th, aged 34. She was the wife of Dr. Frank Pearson and besides him she leaves a son and two daughters. Born in Winnipeg, she was educated there, graduating in Medicine in 1949. She was a member of St. Margaret's Church, Winnipeg, and of the Professional and Business Women's Club in St. James.

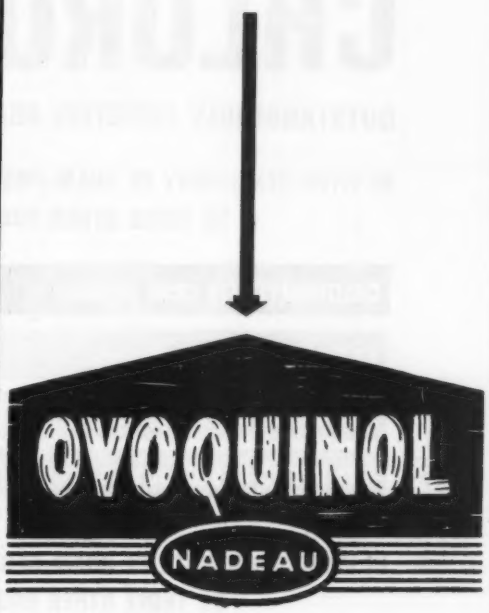
☆

Dr. Frank Smith Macdonald

Dr. Frank Smith Macdonald, 50, died suddenly at his home in Winnipeg on March 31st. Born in Scotland he came with his family to Winnipeg where he was educated in the city schools and graduated in medicine in 1934. He practised in Spalding, Man., Kindersley and Saskatoon, Sask., McBride, B.C. and in Winnipeg. He served overseas with the 8th Army medical corps in Europe 1943-45. He is survived by his wife, two daughters, a brother and two sisters.

LEUKORRHEA

VAGINAL INFECTIONS



CONES AND TABLETS

TWO FORMULAS

OVOQUINOL-PLAIN

	Cones	Tab.
Diiodohydroxyquinoline U.S.P.	75 mg.	75 mg.
Sodium Propionate	500 mg.	250 mg.
Sulfadiazine U.S.P.	400 mg.	400 mg.
Phenoxyethanol B.P.C.	0.04 ml.	
Destrose and Lactose	q.s.	q.s.

OVOQUINOL-OESTRO

Same formula as OVOQUINOL-PLAIN plus 0.01 mg. Ethinyl-oestradiol B.P. per cone or tablet.

DOSAGE

One or two cones or tablets per day preferably at bed-time or as prescribed by the physician.

NADEAU LABORATORY LTD.

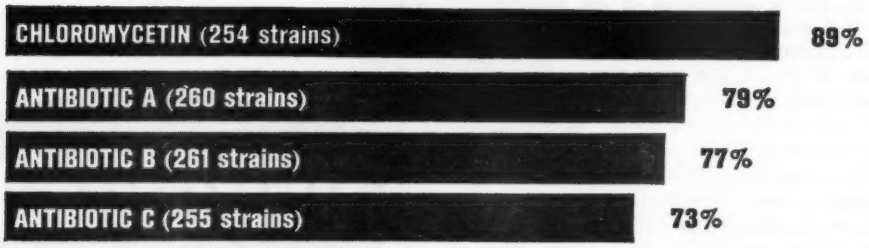
Montreal Canada

True broad-spectrum coverage...
proved clinical efficacy

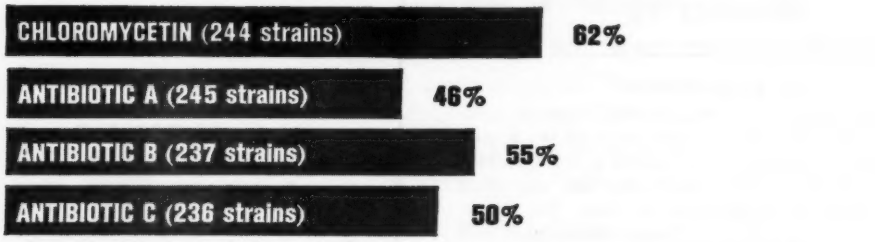
CHLOROMYCETIN®

OUTSTANDINGLY EFFECTIVE AGAINST A WIDE RANGE OF PATHOGENS

IN VITRO SENSITIVITY OF GRAM-POSITIVE ORGANISMS TO CHLOROMYCETIN AND TO THREE OTHER BROAD-SPECTRUM ANTIBIOTICS*



IN VITRO SENSITIVITY OF GRAM-NEGATIVE ORGANISMS TO CHLOROMYCETIN AND TO THREE OTHER BROAD-SPECTRUM ANTIBIOTICS*



*Adapted from Leming, B. H., Jr., & Flanigan, C., Jr., in Welch, H., & Marti-Ibañez, F.: Antibiotics Annual 1958-1959, New York, Medical Encyclopedia, Inc., 1959, p. 414.

CHLOROMYCETIN (chloramphenicol, Parke-Davis) is available in various forms, including Kapseals® of 250 mg., in bottles of 16 and 100.

CHLOROMYCETIN is a potent therapeutic agent and, because certain blood dyscrasias have been associated with its administration, it should not be used indiscriminately or for minor infections. Furthermore, as with certain other drugs, adequate blood studies should be made when the patient requires prolonged or intermittent therapy. 03760

PARKE-DAVIS **PARKE, DAVIS & CO., LTD.** · **MONTREAL 9, P. Q.**

®REG. TRADEMARK

HOSPITAL MEETINGS

Hospital	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
Children's	12:00 Surgical Rounds	10:00 Staff Rounds "B" Service 12:00 Postgraduate Seminars	9:30 Staff Rounds "C" Service 11:00 Clinical Path. Conference 11:00 Death Review (4th Wednesday)	11:00 Grand Ward Rounds	10:00 Staff Rounds "A" Service	9:00 Newborn Conference
Deer Lodge	Clinical Luncheon (1st Monday)			11:00 Ward Rounds		
Grace		Clinical Luncheon (3rd Tuesday)	9:00 Paediatrics 9:45 Medicine 10:30 Ob. & Gyn. 11:15 Surgery	12:00 - 2:00 p.m. Weekly Seminar		
Misericordia	1. Dept. of Surgery 2. Medical Records 3. Tissue Committee 4. Dept. of Medicine	2. Clinical Luncheon 3. Dept. Obs. & Gyn. 4. Dept. Gen. Prac.	2. Interne Committee			8:30-12:30 Ward Rounds
Municipal			7:30 p.m. Review of Deaths (2nd Wednesday)			8:30 Clinical Staff Conf. and Ward Rounds
St. Boniface	11:00 Paediatric Rounds	11:00 Surgical Rounds 11:00 Obstetrical Rounds	11:00 Grand Rounds 11:00 Cardio-Pulmonary Conf. (2nd Wednesday)	3. 8:00 Orthopedic Rounds 11:00 Tumor Clinic 12.00 Clinical Luncheon (2nd & 4th Thurs.	11:00 Medical Rounds 11:00 Cardio-Pulmonary Conf. (4th Friday)	
St. Boniface Sanatorium		12:30 Clinical Luncheon (2nd Tuesday)				
Victoria		Tissue Committee (1st Tuesday)			1. Death Review 3. Clinical Luncheon 4. Active Med. Staff	
Winnipeg General		11-12:00 Medical Ward Rounds	9-10:00 Tumor Clinic 10-11:00 Surgical	12:15 Clin. Lunch. (1st & 3rd Thurs.)		
Brandon General			Medical Staff Lunch (Wed. prior to 2nd Tues. each month)			10:00-noon Clinical Drs. from S.W. Manitoba invited (Nov. to May)

BUTIBEL®

antispasmodic-sedative

puts the "jumpy," nervous g. i. tract back on schedule—with its regulative antispasmodic-sedative action.

BUTIBEL brings relief through the non-cumulative sedation of 15 mg. BUTISOL Sodium® butabarbital sodium combined with the antispasmodic action of natural extract of belladonna 15 mg. (per tablet or 5 cc.)—each ingredient having approximately the same duration of action.

BUTIBEL Tablets • Elixir • Prestabs® Butibel R-A
(Repeat Action Tablets)

McNEIL

McNeil Laboratories of Canada Limited
357 College Street, Toronto, Ontario